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CONTENTS OF VOLUME 4

JULY, 1926. NUMBER 1

PAGE

Displacement Irrigation of Nasal Sinuses: A New Procedure in Diagnosis and Conservative Treatment. Arthur W. Proetz, M.D., St. Louis... 1

Mixed Salivary Tumor in the Tonsillar Region. A. O. Freedman, M.D., Montreal 14

The Medicolegal Importance of Microscopic Investigation of the Temporal Bone. Clyde A. Heatly, M.D., Rochester, N. Y..... 28

Otologic Complications of Basal Skull Fracture. C. F. Yerger, M.D., Chicago 35

Aphonia: The Report of Three Cases. W. R. Watson, M.D., Philadelphia 46

Comparison of the Results Made with Two Types of Audiometer. Harvey Fletcher, Ph.D., New York..... 51

Bilateral Jugular Resection for Bilateral Sigmoid Sinus Thrombosis (Otitic): Report of Cases. Hill Hastings, M.D., Los Angeles..... 58

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology. Intradural Complications of Aural and Nasal Origin: A Survey of Recent Literature. Wells P. Eagleton, M.D., Newark, N. J..... 69

Abstracts from Current Literature..... 82

Society Transactions:

College of Physicians of Philadelphia, Section on Otolaryngology... 93

AUGUST, 1926. NUMBER 2

Bacteremia Associated with Acute Hemolytic Streptococcic Throat Infections. Howard C. Ballenger, M.D., Winnetka, Ill..... 97

Hysterical Dysphagia. Herman J. Moersch, M.D., and H. Milton Conner, M.D., Rochester, Minn..... 112

Cultures from Tonsils. Charles W. Richardson, M.D., Washington, D. C... 120

Bilateral Paralysis of the Adductors of the Vocal Cords of Organic Origin: Report of a Case of Landry's Paralysis. Paul B. MacCready, M.D., New Haven, Conn..... 122

The Occurrence of Otosclerosis in the Etiology of Progressive Deafness. George E. Shambaugh, M.D., and Jacob W. Holderman, M.D., Chicago 127

Reactions Induced Through the Ductus Endolymphaticus and the Aqueductus Cochleae. Lester Mead Hubby, M.D., New York..... 137

Results of Tonsil Operations: A Questionnaire Report. F. J. Pratt, M.D., Minneapolis 142

Carcinoma of the Larynx and Laryngopharynx Treated with Radium: An Analysis of Thirty Cases. Charles J. Imperatori, M.D., New York... 151

Clinical Notes:

The Audio-Controller. O. C. Smith, Jacksonville, Ill., with a Preface by George E. Shambaugh, M.D., Chicago..... 160

AUGUST—Continued

PAGE

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology. Intradural Complications of Aural and Nasal Origin: A Survey of Recent Literature (Concluded). Wells P. Eagleton, M.D., Newark, N. J. 163

Abstracts from Current Literature 176

Society Transactions:

Joint Meeting of the Section on Otology and Laryngology of the New York Academy of Medicine, and the College of Physicians of Philadelphia, Section on Otology and Laryngology 186

SEPTEMBER, 1926. NUMBER 3

Pathology and Surgery of the Eustachian Tube, with Special Reference to the Value of Closing It. Thomas H. Halsted, M.D., Syracuse, N. Y.. 189

Local Treatment of the Eustachian Tube, with Especial Reference to the Present Status of the Bougie. Arthur B. Duel, M.D., New York.... 196

Malignant Tumors of the Antrum of Highmore: End-Results of Treatments. Gordon B. New, M.D., Rochester, Minn 201

Radium in Polypoid Ethmoiditis: Report of Cases. Samuel McCullagh, M.D., and G. Allen Robinson, M.D., New York 215

Iodized Oil in the Diagnosis of Nasal Sinus Conditions. Henry M. Goodyear, M.D., Cincinnati 223

The Interpretation of Speech by Tactual and Visual Impression. Robert H. Gault, Ph.D., Washington, D. C 228

Clinical Note:

Ephedrine in Nasal Disturbances. J. W. Davies, M.D., New York... 240

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology. Acute and Chronic Otitis Media and Sinus Thrombosis. Samuel J. Kopetzky, M. D., New York 241

Abstracts from Current Literature 254

Society Transactions:

American Laryngological, Rhinological and Otological Society 264

OCTOBER, 1926. NUMBER 4

Ablation Experiments on the Labyrinth of Frogs. John Tait, M.D., Montreal 281

Postadenoidal Speech. John A. Glassburg, M.D., New York 296

Typhoid Fever Simulating Sigmoid Sinus Thrombosis. O. Jason Dixon, M.D., Kansas City, Mo 298

Zygomatic Mastoiditis Without Clinical Otitis Media. M. L. Breitstein, M.D., Baltimore 300

Phenobarbital in the Prophylaxis and Treatment of Acute Cocaine Intoxications: A Preliminary Report of Its Clinical Trial. M. Reese Guttman, M.D., Chicago 304

An Easier Method of Introducing the Esophagoscope. M. Joseph Mandelbaum, M.D., New York 307

Pathology of Spheno-Ethmoidal Sinusitis. Bernard J. McMahon, M.D., M.Sc. (Med.), St. Louis 310

OCTOBER—Continued

PAGE

Clinical Notes:

Extensive Lung Abscess Involving the Entire Right Inferior Lobe: Report of a Case Treated Successfullly with Suction Through the Bronchoscope. Joseph W. Miller, M.D., New York 334

A New Tonsil Needle Holder and Artery Hemostat. Morris A. Weinstein, M.D., Philadelphia 338

A New Procedure for the Closure of Nonspecific Perforations of the Nasal Septum. M. A. Lischkoff, M.D., and C. J. Heinberg, M.D., Pensacola, Fla 342

Abstracts from Current Literature 343

Society Transactions:

Chicago Laryngological and Otological Society 357

Book Reviews 374

NOVEMBER, 1926. NUMBER 5

External Operation on the Ethmosphenoid-Frontal Group of Sinuses Under Local Anesthesia: Technic for Removal of Part of Optic Foramen Wall for Relief of Pressure on Optic Nerve. Edward Cecil Sewall, M.D., San Francisco 377

Two Anatomic Preparations of the Temporal Bone for Teaching Purposes. G. E. Tremble, M.D., Montreal 412

Tonsillectomy by Nerve Block Anesthesia. Francis V. Gowen, M.D., Philadelphia 421

Clinical Notes:

A Special Syringe for Displacement Irrigation of the Nasal Sinuses. Arthur W. Proetz, M.D., St. Louis 424

Tonsillectomy with the Tonsillectome. M. C. Myerson, M.D., Brooklyn 425

Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology: Plastic Surgery. J. Eastman Sheehan, M.D., New York 427

Advancement in Our Knowledge of Allergy as Related to Otolaryngology During the Past Two Years. W. W. Duke, M.D., Kansas City, Mo.. 430

News and Comment:

National Society Meetings 445

Local Society Meetings 445

Abstracts from Current Literature 446

Society Transactions:

Chicago Laryngological and Otological Society 458

American Otological Society 459

Book Reviews 477

DECEMBER, 1926. NUMBER 6

Tuberculosis of the Larynx. Treatment by Surgical Intervention in the Superior and Inferior Laryngeal (Recurrent) Nerve: A Report Based on Seventy-Nine Cases. Henry P. Schugt, M.D., New York 479

Nonendoscopic Cases of Foreign Bodies in the Tonsil. Louis H. Clerf, M.D., Philadelphia 489

Contractured Ulnar Paralysis Relieved by Treatment of the Nasal (Sphenopalatine) Ganglion. W. R. Brandon, M.D., New York 493

DECEMBER—Continued

PAGE

The Thymus Gland. W. Walter Wasson, M.D., Denver 495

Acute Suppurative Mediastinitis: Report of a Case. Fred E. Ball, M.D., Chicago 512

Erosion of the Petrous Bone by Acoustic Nerve Tumor: Demonstration by Roentgen Ray. E. B. Towne, M.D., San Francisco 515

Clinical Notes:

A Needle in the Cervical Region Tolerated for Five Months. J. N. Roy, M.D., Montreal 520

A New Procedure in the Treatment of Chronic Maxillary Sinus Suppuration in Children. Arthur M. Alden, M.D., St. Louis 521

An Improved Aural Speculum. George L. Richards, M.D., Fall River, Mass. 525

Progress in Otolarygology: Summaries of the Bibliographic Material Available in the Field of Otolaryngology. Tuberculosis of the Throat. George B. Wood, M.D., Philadelphia 527

News and Comment 536

Abstracts from Current Literature 538

Society Transactions:

American Laryngological Association 551

Book Reviews 568

General Index 569

JUL 21 1926

VOLUME 4 NUMBER 1

ARCHIVES OF
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EDITORIAL BOARD

GEORGE E. SHAMBAUGH, Chicago
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CONTENTS OF PREVIOUS NUMBER

JUNE, 1926. NUMBER 6

The Chemotherapy and Serum Therapy of Pneumococcus and Streptococcus Meningitis. John A. Kolmer, M.D., Philadelphia.

Some Phases of Streptococcic Infection in Acute Mastoiditis. Richmond McKinney, M.D., Memphis, Tenn.

Postdiphtheritic Stenosis of the Larynx. Henry Boylan Orton, M.D., Newark, N. J.

Clinical Notes:

The Improved Endoscope for Closing Safety-Pins. A. J. Shekter, M.D., Jamaica, L. I.

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology. Chronic Progressive Deafness, Including Otosclerosis and Pathology of the Inner Ear. J. K. M. Dickie, Ottawa, Ont.

Correspondence:

Functional Examination of Hearing. J. Ivimey Dowling, M.D., Albany, N. Y.
Reply of Robert Sonnenschein, M.D., Chicago.

News and Comment:

Local Society Meetings.

Abstracts from Current Literature.

Society Transactions:

Chicago Laryngological and Otological Society.

Book Review.

Index.

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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 4 JULY, 1926 NUMBER 1

DISPLACEMENT IRRIGATION OF NASAL SINUSES

A NEW PROCEDURE IN DIAGNOSIS AND CONSERVATIVE TREATMENT *

ARTHUR W. PROETZ, M.D.

ST. LOUIS

The procedure here presented is a simple method of introducing fluids into the sphenoid and posterior ethmoid sinuses. Heretofore, the irrigation of these sinuses has been rendered difficult by their positions and the inaccessibility of their ostia. Only occasionally can the normal opening of the sphenoid be used for the introduction of a cannula, while in the case of the posterior ethmoid cells it is practically impossible. Perforating the sinus walls, for the purpose of instilling fluids, is dangerous; it is not always accurate, and it produces inflammatory reactions which defeat the essential purpose of the treatment.

It occurred to me that those very mechanical obstacles which prevent the proper drainage of the posterior cells might be turned to account in similarly retaining therapeutic solutions in contact with the lining membrane. My problem, therefore, was the introduction of such solutions into the sinuses without producing trauma, and the following method was devised:

METHOD

1. The patient is placed in the supine position, with his head projecting beyond the top of the treatment chair or table. The occipito-atloid joint is extended until the tip of the chin and the external auditory meati are in the same vertical plane. This renders the sphenoid sinus, with its ostium upturned, the most dependent structure in the nasal chamber. A "V" shaped pocket thus occurs at the junction of the face of the sphenoid with the cribriform plate of the ethmoid.

2. The fluid to be introduced is now allowed to flow into the nostrils from a syringe and comes to rest in this pocket, submerging the ostia of the posterior sinuses. It does not enter the sinuses, as their ostia are of insufficient size to permit the escape of the contained air.

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* From the Department of Otolaryngology, Washington University School of Medicine.

* Read before the Section on Laryngology, Otology and Rhinology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

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3. Gentle suction (not more than three pounds) is now applied intermittently to one nostril, the other being closed, the palate and tongue being held in the "K" position to seal the pharynx. Any of the ordinary vacuum appliances will suit, provided that the degree of tension can be controlled. When the suction is first applied, a bubble of air escapes from the sinus through its upturned ostium. On releasing the suction this air is replaced by a drop of the overlying fluid. The process is repeated until the sinus is full; about a dozen alternations usually suffice to accomplish this.

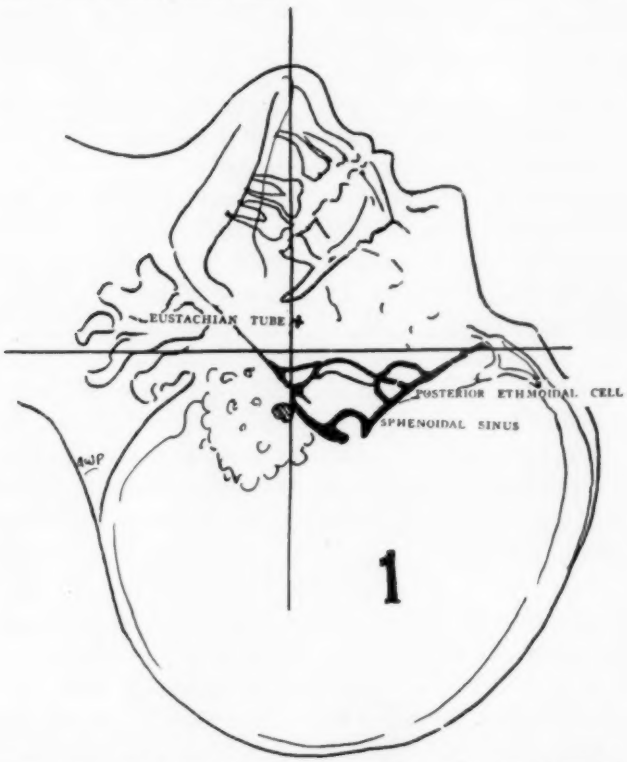

Fig. 1.—Diagram indicating the correct position of the head for displacement irrigation. The parts immediately concerned are heavily outlined. This diagram corresponds to the roentgen-ray plate 1, in each case.

4. The patient is returned to the erect position, and the fluid remains in situ for an indefinite period (eight hours to several days).

In practice, I let down the back of the small treatment chair, and, sitting on a low stool behind the patient, rest his head on my knees. The position is thus readily controlled. The nurse is ready with the loaded syringe and the suction tip, and the whole procedure occupies less than a minute. It is often of advantage to shrink the membrane with a mild astringent before instituting the treatment.

The first obstacle encountered proved to be more apparent than real. This was the possible danger of accidentally introducing fluids into the middle ear through the eustachian tube. It was found that with the head in the position described, the nasal chamber could be filled to the level of the then uppermost ostium, that of the maxillary sinus, without approaching within 1 cm. of the tubal opening. But a fortunate anatomic situation renders precaution in this regard unnecessary, namely, the length and character of the tube. The low vacuum

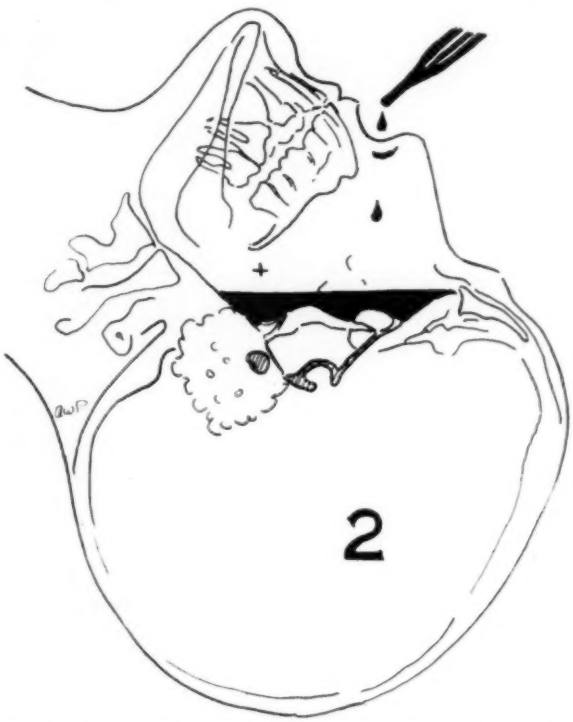

Fig. 2.—Second step. The introduction of fluid into the nostril. It comes to rest in the nasal chamber but does not enter the sinuses.

employed is insufficient to extract a bubble of air from the middle ear, hence no displacement occurs, and the nose may be filled with fluid without any danger of introducing it into the middle ear by this method.

TREATMENT

All cases enumerated in this report were treated with physiologic sodium chloride solution. I purposely refrained from employing other fluids at first, in order to determine the effect of simple mechanical cleansing and of the thinning of viscous exudates retained in the sinuses.

Good results have followed this procedure in most cases. The effect of introducing various astringents, antiseptic solutions and ferments will be reported in a subsequent paper.

The mere dilution of the retained secretions and the clearing of the ostia have resulted in great improvement in many cases. The membrane has been enabled to rid itself of the infection without further treatment. The first of these cases were treated seven months ago, and many of them have remained free from trouble for that period, although epi-

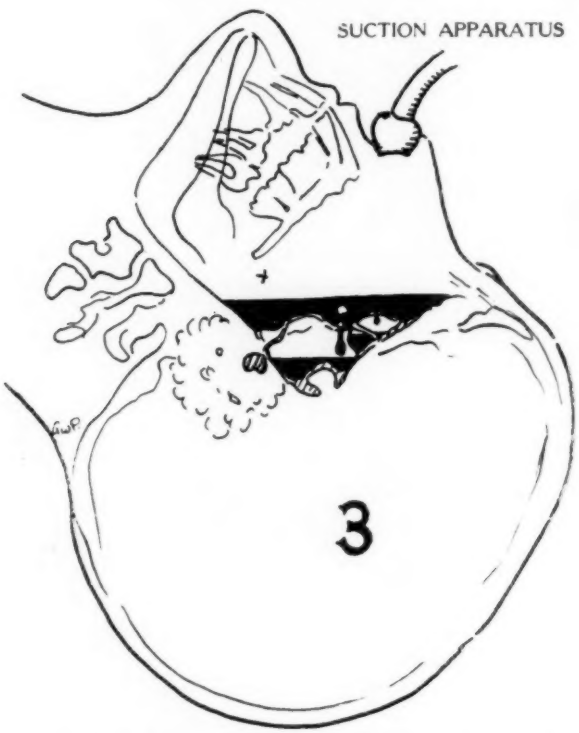

Fig. 3.—Third step. Intermittent suction is applied at the nostril. Air bubbles are withdrawn from the cells, and are replaced by drops of the overlying fluid. Sphenoid and posterior ethmoid cells are shown filling separately.

demics of severe upper respiratory infections have occurred during the intervening months. In four cases a reaction characterized by acute coryza and some headache followed the first irrigation. Three of these cases were irrigated on subsequent occasions without trouble. The fourth case was not irrigated a second time. The treatments are given at intervals of three to eight days, depending on the amount and character of the discharge in the individual case.

DIAGNOSIS

The diagnostic possibilities of this procedure were startlingly revealed when iodized oils were employed to determine the distribution of the fluids in the nose, by means of the roentgen ray. For purposes of study, four exposures were made in each case, corresponding to the steps in the process enumerated above, namely:

Plate 1. The head of the patient in the supine position without oil, for the comparison of landmarks. The chin in a vertical plane with the external auditory meati.

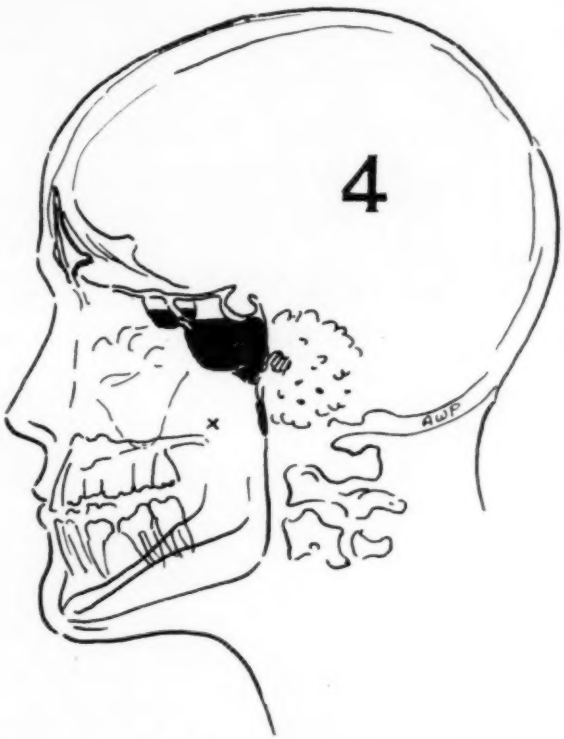

Fig. 4.—Patient returned to the upright position. The fluid remains in the sinuses.

Plate 2. Same position, after the introduction of the oil into the nasal chambers, but before the application of suction.

Plate 3. Same position, after intermittent suction.

Plate 4. Patient returned to upright position (stereoscopic if possible).

The accompanying photographs demonstrate the extent to which 5 cc. of oil in each case entered the sinuses.

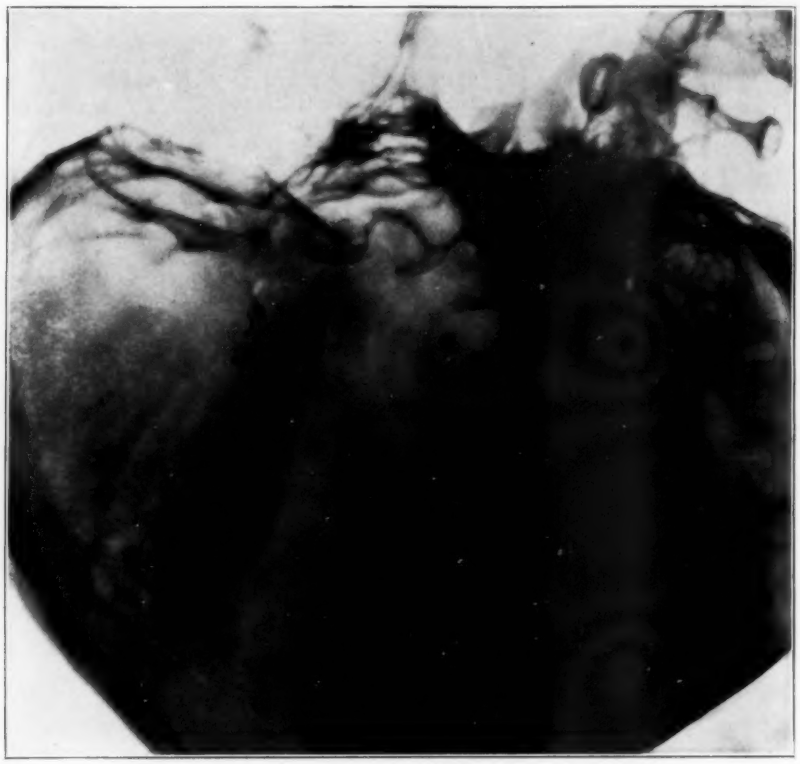

Fig. 5 (case 13).—Miss H. F., plate 1. Control plate.

Fig. 6 (case 13).—Plate 2. Oil introduced into nose, resting in nasal chamber. Suction has not yet been applied. Note absence of oil in the sphenoid.

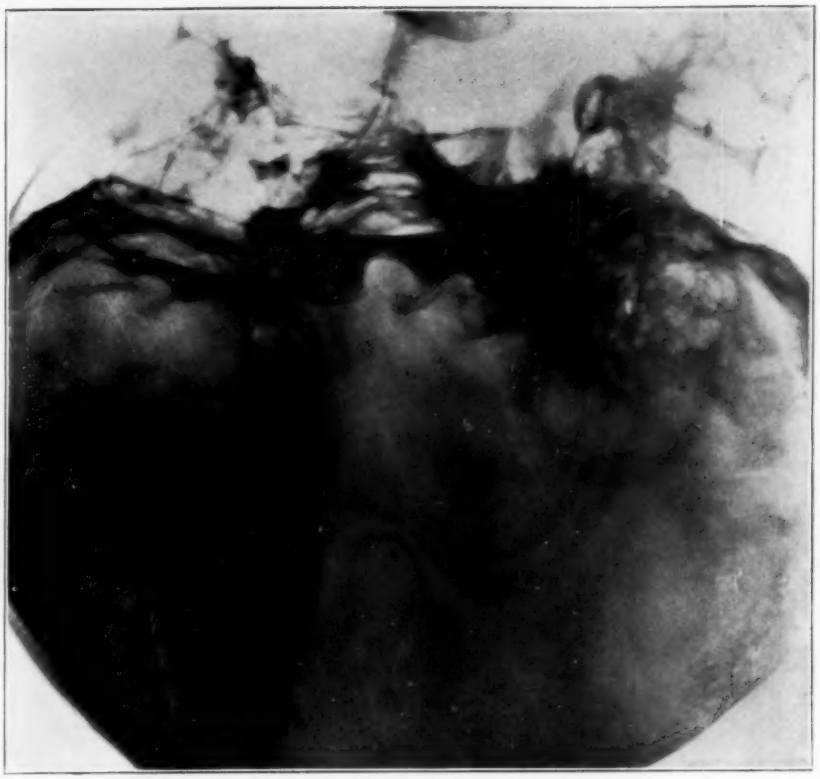

Fig. 7 (case 13).—Plate 3, after application of intermittent suction. Oil is seen in sphenoid and posterior ethmoid cells. A few scattered drops may be seen remaining in the position seen in plate 2.

Fig. 8 (case 13).—Plate 4. Erect position showing the oil in the posterior ethmoid and sphenoid cells.

Fig. 9 (case 1).—Mrs. H., plate 3. Faulty position. The chin was not over the auditory meatus. The fluid occupied the nasopharynx and no penetration was accomplished.

Fig. 10 (case 11).—M. C., plate 3. Oil in sphenoid. The thin white line between the bone of the sella turcica and the oil indicates a thickened membrane as does the space on the right (floor of the sphenoidal cell). This was verified at operation.

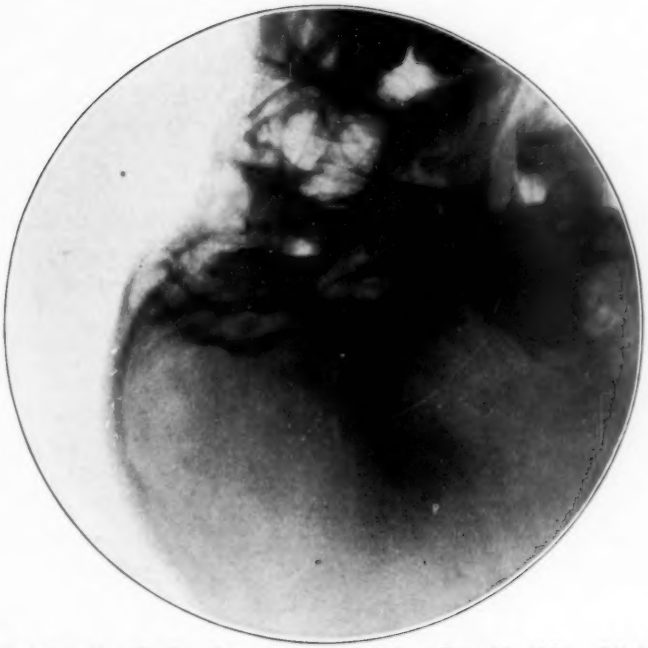

Fig. 11 (case 6).—L. B., plate 3, series 1, taken Jan. 12, 1926. Oil has penetrated posterior ethmoid but has not entered sphenoid.

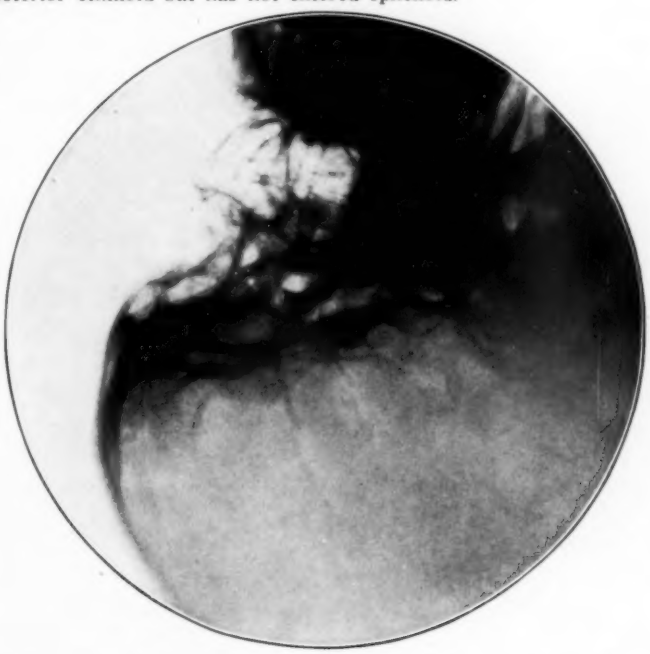

Fig. 12 (case 6).—Series 2, taken February 19 (five weeks later than series 1). The oil is now found in the sphenoids as well as in the posterior ethmoids. The treatment in the interval consisted of four displacement irrigations given one week apart. The headaches and vomiting from which the patient sought relief have not recurred.

It has been found that a better appreciation of the anatomy may be had by only partly filling the cells, as by so doing the fluid level may be recognized in each cell, which assists in identifying and distinguishing overlapping cells.

It will be readily seen that thickened and polypoid membrane within a cell may be detected by discrepancies between the oil shadow and the bone shadow which they occasion. This is shown in case 11 (fig. 10), in which the diagnosis has since been verified at operation.

Several cases have been encountered in which the oil has entered the posterior ethmoid cells but has failed to penetrate into the sphenoid, and vice versa. This may be interpreted in one of two ways: it may occur that the ostia of some cells are patent and that others are not, and that those which are obstructed are sufficiently blocked to prevent the exhaustion of air and the admission of oil. The second possibility is that the sinus is already filled with the products of inflammation and that displacement is therefore not accomplished. In any case, the cells which fail to fill with fluid are abnormal cells. The photographs in case 6 (figs. 11 and 12) are of interest in this regard. The first series, taken January 12, show that oil has penetrated into the posterior ethmoid cells, but not into the sphenoid. The second series was taken February 19. In the interval the patient received one irrigation weekly with physiologic sodium chloride solution and no other treatment. The latter plates show the oil again in the posterior ethmoid cells, but this time in the sphenoid cells as well. The severe headache and nausea from which the patient sought relief have disappeared without a recurrence since the latter date.

This treatment is not presented as a panacea. I have always opposed indiscriminate irrigation of any kind in the early stages of nasal infection, and believe that displacement irrigation may do great harm if employed at this time. I have also refrained from using it in patients with arterial hypertension. It will obviously not relieve those cases of sinus disease in which permanent changes have occurred in membrane, periosteum or bone. The treatment of these is essentially surgical. But it should eradicate those infections which have passed the acute stage and which, due to embarrassment of drainage and ventilation, will in time produce such changes; I believe the majority of cases seen in routine practice belong in this class.

SUMMARY

1. A method is presented for introducing fluids into the posterior series of accessory nasal sinuses without producing any trauma.

2. By the use of appropriate solutions, it may be employed as a therapeutic agent.

Treatment and Results in Thirty Cases

Case	Diagnosis	Comment	No. of Treatments	Interval	Chief Complaint	Result
1. Mrs. H.	Subacute sphenoiditis	.....	3	3 days	Postnasal discharge	Recovery
2. Dr. T.	Chronic sphenoiditis	Severe headache followed first irrigation	2	3 days	Severe headache	Treatment not completed
3. Helen G.	Subacute pansinusitis	Shrinking alone failed to improve	3	3 days	Occipital headache	Recovery
4. E. S.	Chronic pansinusitis	Has had several operations	6	Irregular	Copious nasal discharge	Relief from symptoms
5. Dr. C. L. D.	Subacute sphenoiditis	Later had bilateral sphenoid operation	8 ?	Daily	Failing vision	Vision improved
6. Leitha B.	Chronic sphenoiditis	Headaches occurred every few days	4	1 week	Headache and vomiting frequent and severe	No recurrence since 2/19/26
7. Frank D.	Chronic pansinusitis	Has had several operations	5	Varying	Copious nasal discharge	Relief from symptoms
8. Lydia H.	Atrophic rhinitis	Advanced case	4	Varying	Nasal obstruction and discomfort	Relief from symptoms
9. Mr. G. G.	Chronic sphenoiditis, chronic tonsillitis	Symptoms only partly relieved by tonsillectomy	5	4 to 6 days	Joint pains at frequent intervals	Complete relief
10. Mrs. E. L. H.	Subacute sphenoiditis	Five weeks' duration	4	3 days	Postnasal discharge, headache	Recovery
11. Miss M. C.	Chronic sphenoiditis	No treatment by this method; diagnosis only	1	.....	Multiple arthritis	Diagnosed thickened membrane in sphenoid
12. Ed. C.	Chronic pansinusitis	Has had several operations	3	Varying	Nasal discharge	Immediate relief of symptoms on three occasions
13. Helen F.	Chronic sphenoiditis, tubal catarrh	Severe headache and coryza followed first irrigation	4	1 week	Nasal discomfort, partial deafness	Improvement of nasal condition and of hearing
14. Miriam H.	Subacute sphenoiditis and posterior ethmoiditis	.....	5	5 days	Postnasal discharge	Complete relief
15. Mrs. T.	Chronic sphenoiditis and posterior ethmoiditis	Nasal trouble, 3 years duration; also has pyelitis	6	4 to 6 days	Postnasal discharge	Pronounced improvement in nasal condition; pyelitis not improved
16. Mrs. P. P.	Subacute simple (?) rhinitis	Some deformity both middle turbinates following former operation	4	3 days	Nasal obstruction	Recovery
17. Mrs. W.	Acute posterior group sinusitis	Irrigated only after acute stage subsided	3	3 days	Severe headache	Recovery
18. Miss L.	Acute right sphenoiditis; right maxillary sinusitis	Irrigated only after acute stage subsided	4	2 days	Severe headache	No relief until maxillary sinus was opened and drained
19. Miss LeS.	Subacute posterior ethmoiditis on the left	.....	2	2 days	Painful joints	Went to operation; unsuitable for irrigation
20. Dr. G.	Mild subacute pansinusitis	Three weeks' duration	1	.....	Persistent nasal discharge	Discharge ceased 3 days after irrigation
21. Miss McD.	Atrophic rhinitis	Several operations	3	5 days	Nasal discomfort	Relief questionable
22. Mrs. E. P. H.	Chronic sphenoiditis, bilat.	Irrigated for diagnosis only	1	.....	Sluder's "lower half" headache	Operation
23. Mr. T.	Chronic posterior sinus disease	Severe headache after first irrigation	2	5 days	Severe headache	No relief; discontinued for the present
24. Catherine B	Chronic posterior sinus disease, tubal catarrh	.....	5	3 days	Partial deafness	No improvement
25. Dorothy B.	Atrophic rhinitis, pharyngitis sicca	.....	4	1 week	Dry throat	No improvement to date
26. Mrs. E. S.	Mild subacute sphenoiditis	Six weeks' duration	3	2 days	Postnasal discomfort	Relief
27. Dr. P.	Subacute sphenoiditis	Staph. aureus infection	1	.....	Postnasal discharge	Recovery
28. I. J. S.	Mild chronic pansinusitis	Years' duration	4	3 to 6 days	Frequent temporal and occipital headache	Headaches relieved, heavy sensation in temples remains
29. W. H. K.	Subacute sphenoiditis and posterior ethmoiditis	.....	2	2 days	Postnasal discharge	Improved (incomplete)
30. Bertha H.	Atrophic rhinitis, severe	.....	?	Varying	Nasal obstruction	No improvement

3. By the use of radiopaque oils it may be used in combination with roentgen rays for diagnosis.

4. It is not a substitute for surgical measures in those cases in which extensive permanent tissue changes have taken place, but may prevent such changes if instituted before they have occurred.

234 University Club Building.

ABSTRACT OF DISCUSSION

Dr. George M. Coates, Philadelphia: Dr. Harmon Smith's work, fifteen years ago, was on entirely different grounds, although his hope was to accomplish the same thing. Dr. Lewis A. Coffin's method is the same as that of Dr. Smith, except that vapor is used instead of liquid. Dr. Proetz' method is original, simple and practical. Its value can be determined only by trial. Dr. Proetz shows its value in determining hyperplastic areas in the sinuses that can be distinguished by difference in density between the shadow of the oil and the true bone shadow. As a therapeutic measure, we all agree that medicaments, antiseptics and astringents do good in the nose; they should do equally well in the sinuses.

Dr. Edwin C. Ernst, St. Louis: In Dr. Proetz' work, inflammatory and thickened mucous membrane changes, displacement of the lipiodol shadow, polypoid filling defects, or a partial or complete absence of the lipiodol within the sphenoid sinuses due to tumors or to other types of infiltrations, all are diagnostic, roentgen-ray findings of value.

The types of sphenoid sinus filling defects must be developed similar to the early gastric and duodenal roentgen-ray studies. Furthermore, the position of the patient may be changed or the direction of the ray through the sphenoid can be angulated so that both sinuses may be outlined in the semilateral direction, one above the other. The superior-inferior, or vertical, direction of the ray may give us additional diagnostic information. The Granger position unquestionably may be an added valuable technical procedure.

The outstanding feature of the method is its simplicity. The patient is first of all placed on the horizontal table, face up. It might be desirable to elevate the hips slightly. The head is then placed over the end of the table so that the lipiodol fluid along the posterior superior margin of the pharynx is at a slightly higher level than the anterior area. One direct lateral radiogram is taken before, and another after, the fluid is in position. Then a third lateral radiogram is made immediately after suction has been applied. The procedure is completed within a few minutes, without any discomfort to the patient.

Dr. Harold M. Hays, New York: I should like to ask Dr. Proetz whether it is possible to get fluid into the antrums in the way outlined. If you can get fluids into the sinuses that way, I should like to try the vaccines about which I spoke at the last meeting of this section.

Dr. Arthur W. Proetz, closing: I am glad that Dr. Coates emphasized the simplicity of this maneuver and the short time required. It takes about a minute.

In the other methods mentioned by Dr. Coates, a vacuum is first created and then fluid or vapor is squirted in. This has two drawbacks: First, a high vacuum is required, producing an irritation and hyperemia; second, much of the fluid does not hit the mark. When lipiodol is employed, one should use as little as possible, so as to prevent its flowing into the intestinal tract and producing iodism. With this upside down method, all the solution goes where it is supposed to go; in any event, if through faulty technic it does not enter the sinus, the patient does not swallow it.

For treatment, it is often advisable to shrink the schneiderian membrane: for diagnosis, this is not so desirable.

I wish to thank Dr. Ernst, not only for his discussion, but also for the effort he has made in furthering this work. Most of the radiographic work was done by him.

In answer to Dr. Hays' question about the antrums: We have succeeded in introducing some of the fluid into the maxillary and frontal sinuses. At present, we are busying ourselves with a method of filling these sinuses without having to fill the posterior group as well, as this takes too much lipiodol. I see no reason why vaccines should not be introduced by this method.

MIXED SALIVARY TUMOR IN THE TONSILLAR REGION*

A. O. FREEDMAN, M.D.

MONTREAL

This case is of interest, as it shows a rather unusual site for a mixed tumor of a salivary gland, as well as some interesting pathologic findings associated with it.

REPORT OF CASE

G. W., a returned soldier, aged 34, came for treatment to the otolaryngologic clinic of the Montreal General Hospital on Nov. 4, 1925.

The history given was that he first noticed a small swelling in the region of his right tonsil in 1919. This gradually grew larger until two years prior to examination, when, because of the discomfort in swallowing and phonation, he sought medical advice in a general hospital. At this time, he stated that an incision had been made into the mass without any benefit some months previously. He was advised, and consented to undergo an operation under a general anesthetic, in order to locate some hidden pocket of pus. No pus was found. After this second operation the swelling was not reduced in size, and the patient was frankly told that the physicians did not know the nature of his condition. In spite of a negative Wassermann reaction, he was given several injections of arsphenamine. This did not have any effect on the tumor.

Since then, no treatment has been employed. The tumor has never been painful; but during the past few months it has increased somewhat rapidly in size, and the discomfort in swallowing and talking has become more marked.

Examination.—This showed a swelling in the region normally occupied by the right tonsil, which bulged into the oral and pharyngeal cavities. Its short axis obliterated more than half of the isthmus faucium. Its long axis was directed anteroposteriorly, and measured about 3½ inches, the anterior pole representing the bulging of the tumor into the mouth, the posterior pole taking up the right lateral pharyngeal wall and projecting into the posterior pharynx as a rounded, smooth projection (fig. 1).

Without this history—judging by the location, size, contour and the patient's nasal voice—one would be tempted to make a snap diagnosis of peritonsillar abscess. On close inspection, however, it was found that the mucous membrane covering the mass was neither red nor angry looking, but rather paler and bluer than the remainder of the mucous membrane of the mouth.

The surface of the tumor was smooth; there was a linear scar on its anterior surface. The mass was not tender to the touch; it was slightly boggy in the center of its anterior surface.

At its lower anterior pole, and posteriorly where it extended into the pharynx, it felt hard and cartilaginous. Because of its situation, it was difficult to say whether it was adherent or not to the neighboring structures. A posterior rhinoscopic examination revealed that the tumor almost obliterated the right posterior choana, obliterated the whole soft palate of that side, but did not quite reach the posterior wall of the pharynx.

There was no evidence of tonsillar tissue on the right side. On the left side there was a normal tonsil in a normal bed. The regional lymph nodes were normal. In order to make a diagnosis, it was decided to remove a portion of the

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*From the Departments of Otolaryngology and Pathology of the Montreal General Hospital.

tumor for microscopic examination. Under local anesthesia, a longitudinal incision was made over the anterior surface of the tumor. When the mucous membrane and the thin palatoglossal muscle was cut through, a white, bloodless capsule presented itself. With a blunt instrument this capsule was shelled out so easily from its bed that the impulse to shell out the whole tumor was too great to be resisted. The incision was elongated. All went well until the lower anterior pole was reached when definite adhesions to the inner surface of the angle of the jaw were found. At this stage, with the forcible endeavor to break down the adhesions

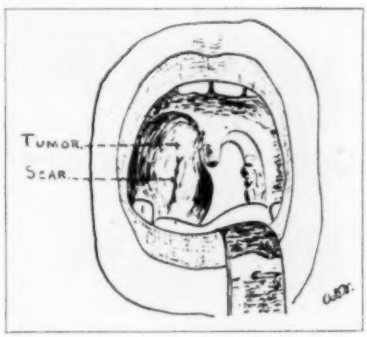

Fig. 1.—Mixed salivary tumor in the right tonsillar region (before operation).

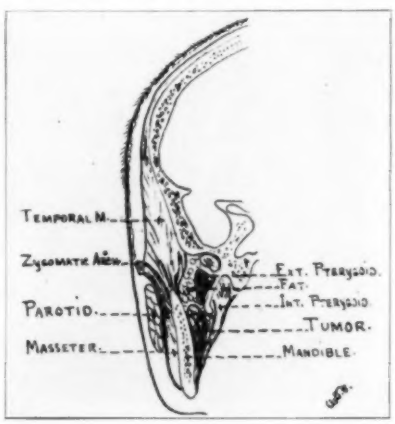

Fig. 2.—Coronal section through the head, showing the relative position of the tumor to the mandible, internal pterygoid and the parotid.

and repeated traction of the tumor, the capsule broke, and a soft gelatinous material appeared. Because of the firm attachment to the lower jaw and the gelatinous nature of the center of the tumor, a tentative diagnosis of sarcoma of the lower jaw was made. A large piece of the tumor was taken away for biopsy. The wound was sutured and a small gauze drain was inserted. A roentgenogram of the jaw was requested.

In a few days the wound healed. The roentgen-ray report read: "A small area of absorption at the angle of the lower jaw in the neighborhood of the last molar tooth."

The microscopic sections showed that the condition was a mixed tumor of the salivary type. This definite information threw an entirely new light on the diagnosis and prognosis, as it is definitely known that tumors of this type do not invade bone. A second series of stereoscopic roentgen-ray pictures were taken; these showed no involvement of bone. Eight days after the first operation, when the wound had completely healed, total removal of the tumor was decided on.

I discussed the case with Dr. Archibald Stewart, who consented to assist me with the operation.

Operation.—Under general anesthesia, intratracheal method, I made a longitudinal incision of 1½ inches on the anterior surface of the tumor, just a little lateral to the first incision. Because of the previous operation, it was a little difficult to get the line of cleavage between the capsule and the rest of the tissues in front, but this was overcome gradually. Behind and above, the tumor shelled

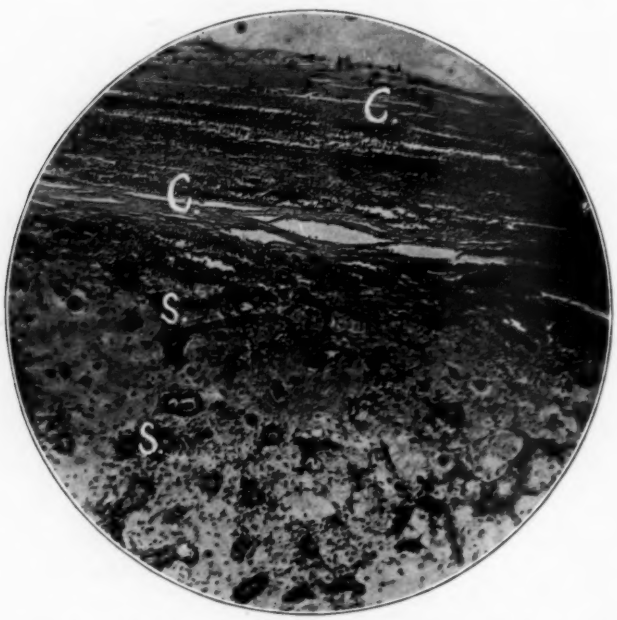

Fig. 3.—Anterior pole of tumor. Capsule, C; solid interlacing strands of cells, S; myxomatous tissue, M. Low power magnification.

out more easily. At the anterior and lower pole, the same difficulty of separating the tumor mass presented itself as in the first operation. But with the knowledge gained from the roentgenograms and the pathologic diagnosis clear, we persisted in patiently "digging" out this pole. After careful dissection and separation, it was found that this part of the tumor lay between the internal pterygoid muscle and the inner surface of the angle of the jaw (fig. 2). The muscle was divided and the lower pole was lifted out of its bed. It was firmly bound down to the periosteum by adhesions, but did not spring from the bone, as we suspected in the first instance. The rest of the tumor shelled out quite easily until we came to the outer surface of the posterior, or pharyngeal, pole, where a second obstruction was met. The anterior part of the tumor was removed in one piece, then the main mass of the tumor was removed in pieces, leaving a large piece posteriorly attached. After separating and dissecting about this part, one could feel the tip

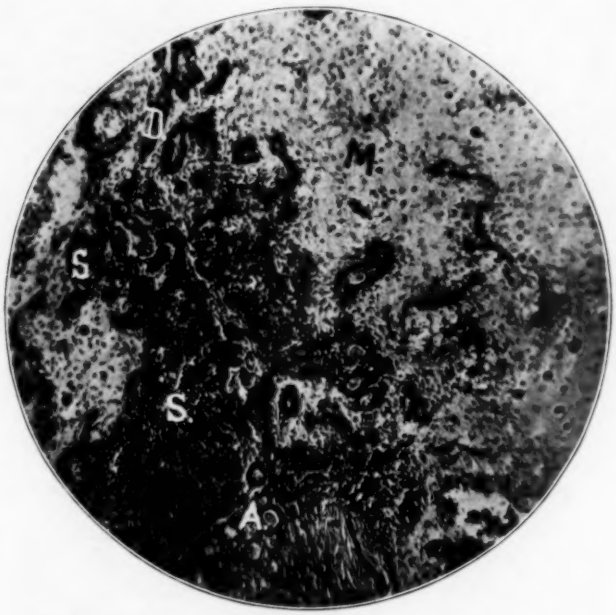

Fig. 4.—Solid masses of cells, S; alveoli, A; elongated ductlike structures, D; myxomatous tissue, M. Low power magnification.

Fig. 5.—Note the polyhedral shape of the cells in the solid strands, S, and the absence of connective tissue between the individual cells. Myxomatous tissue, M. High power magnification of fig. 4.

of the styloid process behind, and the posterior border of the ascending ramus of the mandible in front. A finger could be passed all around a narrow connecting isthmus of tumor. It then became clear that this neck of tumor connected the parotid on the outside with the main mass of tumor on the inside, which had burrowed its way into the fatty space between the internal pterygoid muscle and the inner surface of the lower jaw (fig. 2).

A wire loop was passed round the remainder of the tumor and it was snared off from its attachment to the parotid. A large clean hole now presented itself to the view, bounded behind by the vertebrae, above by the tips of the styloid and mastoid processes, laterally by the sternocleidomastoid muscle and large vessels of the neck, and the inner surface of the angle of jaw as far forward as the last molar tooth. The wound was lightly packed with iodoform gauze; the edges were trimmed and approximated. The whole operation was almost bloodless. The

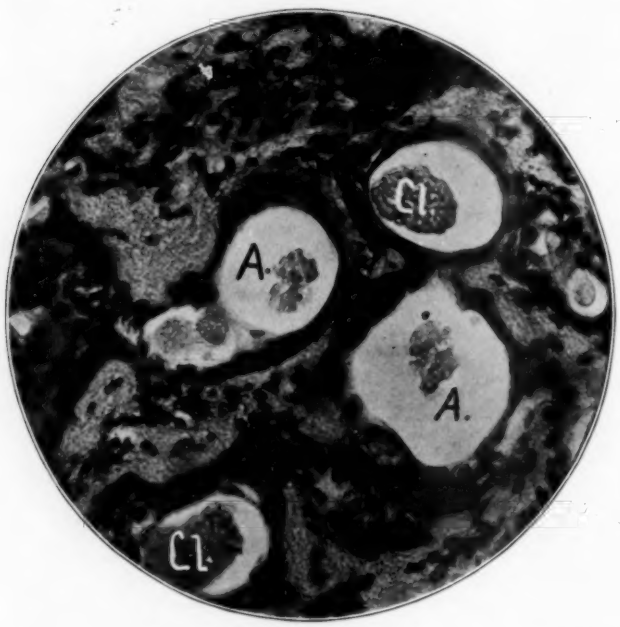

Fig. 6.—Alveoli lined with flattened cells with large oval nuclei, A; colloid, Cl. High power magnification of fig. 4.

patient made an uninterrupted recovery and the wound healed in ten days, leaving a smooth lateral pharyngeal wall.

To obviate possible local recurrences, we propose to follow up this patient with radium treatment, using only the gamma rays to destroy any embryonic cells that might have leaked into the wound during the process of manipulation.

Pathologic Report.—The tumor, which was removed in several pieces, when put together measured 2½ by 3½ inches. A capsule, poorly supplied with blood vessels, varying in thickness from 0.5 to 2 mm., surrounded the whole mass, but because of the difficulty encountered in removal, it was torn in many places.

The cut surface of the tumor, when hardened, was bluish-white and cut like cartilage in some areas; in other areas it was pale yellow, and in still other parts there were areas of softening filled with gelatinous material. At various spots one could see tough fibers of connective tissue crossing in all directions.

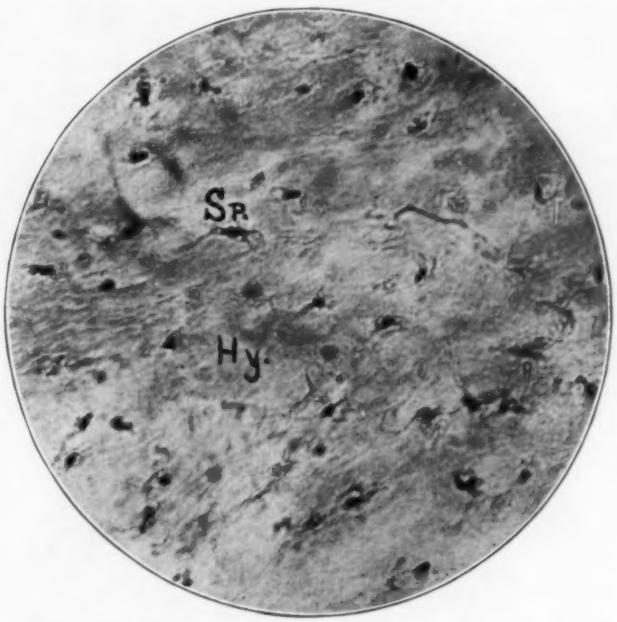

Fig. 7.—Center of tumor, myxomatous stroma. Spindle cells, Sp; hyaline matrix, Hy. High power magnification.

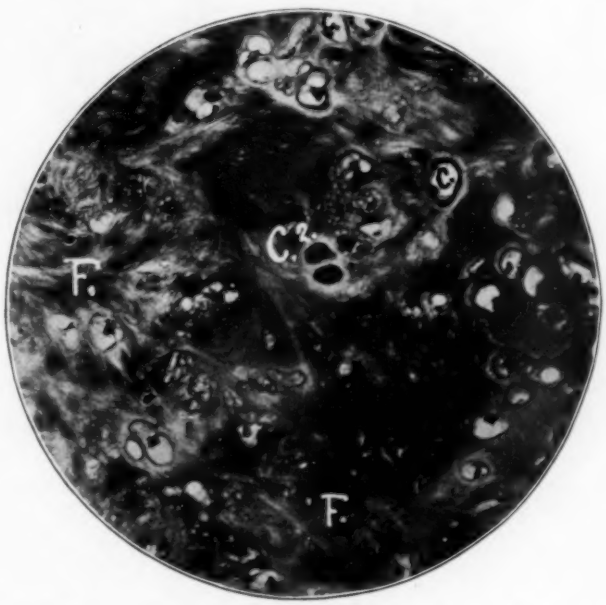

Fig. 8.—Typical section of fibro-elastic cartilage found in nodules all over the tumor. Cartilage lacunae, with shrunken cartilage cells, C; large plump cartilage cell with capsule, C 2; fibrous tissue, F. High power magnification.

Microscopic Findings.—These varied, according to the part of the tumor from which the sections were taken.

The periphery of the tumor shows a distinct capsule. The stroma is made up of loose myxomatous tissue with spindle cells. The parenchyma in this section is arranged in solid strands of cells which communicate with one another by branches of cells (fig. 3).

A section taken from near the center of the tumor shows the solid arrangement of the parenchymal cells. In addition, it shows alveoli, scattered here and there amid the parenchyma. Some of these spaces are duct-like, and are lined with double rows of cells. The alveoli contain a homogeneous colloid substance. The rest of the section is composed of embryonic connective tissue (fig. 4).

A higher power magnification of figure 3 shows that the cells composing the solid strands are polyhedral or oval in shape, have large nuclei, and are solidly

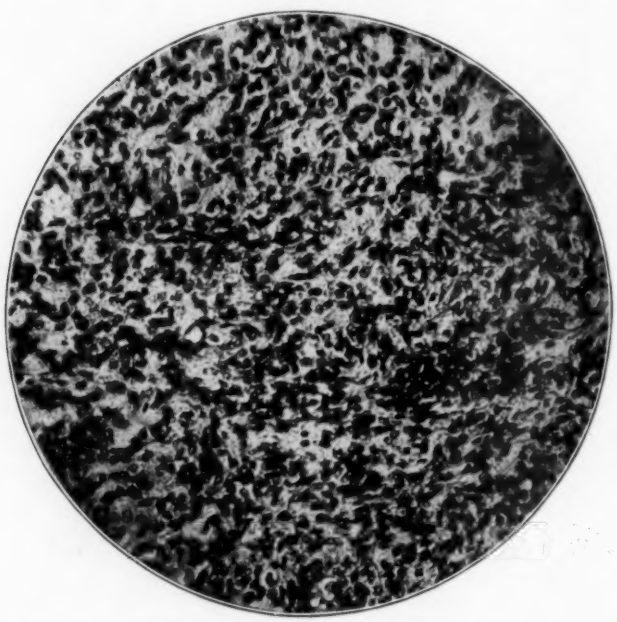

Fig. 9.—Section through pedicle of tumor. Note the homogeneous nature of the cells, and their slight similarity to the cells of the parotid gland.

packed one against the other, without any intervening fibers of connective tissue, thus distinguishing them from sarcomas (fig. 5).

Figure 6 is a higher power magnification of the alveoli in figure 4. It shows that the alveoli are lined by large flat cells, resembling epithelial cells in their grouping; the nuclei are oval and stain diffusely. The alveoli are filled with a homogeneous acidophile staining mucoid material. In other sections the alveoli are lined with a higher columnar epithelium.

Sections taken through the friable pieces which came away from the center of the tumor, show almost a uniform stroma of young myxomatous tissue with the characteristic spindle cells (fig. 7).

Scattered throughout the tumor, nodules of cartilage could be seen and felt, even macroscopically, but the anterior and posterior pole particularly seemed to be composed entirely of cartilage. Figure 8 is a section through part of the anterior

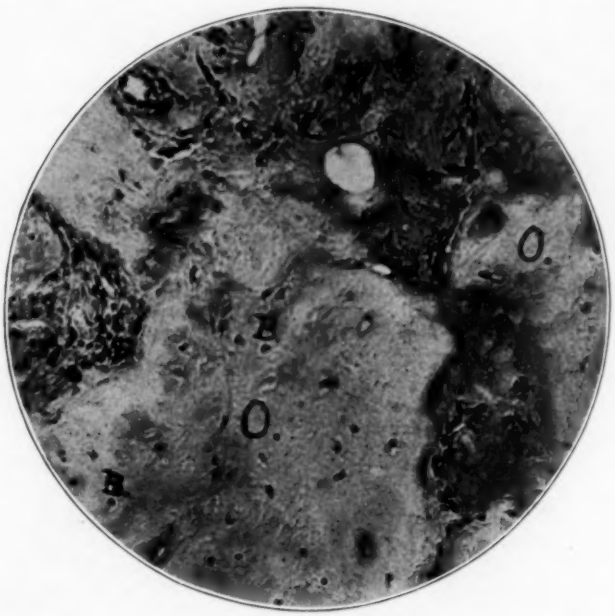

Fig. 10.—Osteoid, O; bone cells in bone lacunae, B. High power magnification.

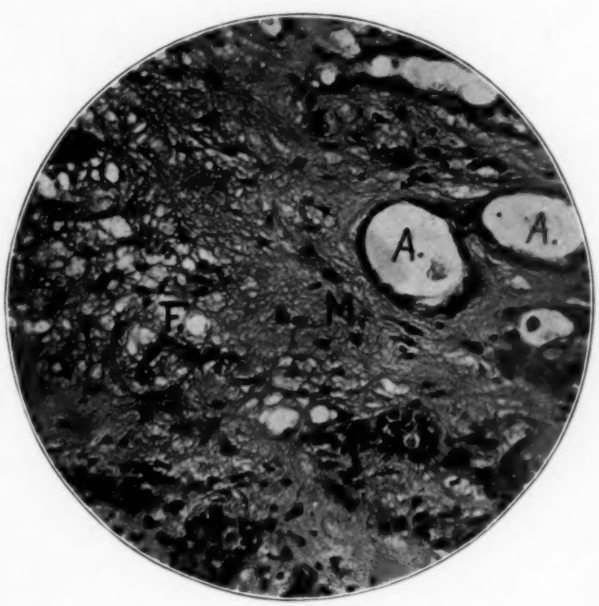

Fig. 11.—Fat cells, F; alveoli, A; myxomatous tissue, M.

pole. Note the coarse bundles of the fibro-elastic tissue running in every direction, the cartilage lacunae with the single and double cartilage cells and the active young cartilage cells in the process of division.

A section through the posterior pole of the tumor where it was snared off from the parotid gland shows a discrete arrangement of round, oval and polygonal cells, with small nuclei. There is very little stroma in this part of the tumor, and it resembles the normal parotid a great deal (fig. 9).

Another section from the broken down center of the tumor shows osteoid formation. The osteoid is eosinophilic, has no calcium deposit and is not lamellated, but has definite bone lacunae and plump bone cells in the lacunae (fig. 10).

In the many sections examined, fat was only found in small areas (fig. 11). The alveoli in this section are lined with flat cells which have been mistaken for endothelium by some observers.

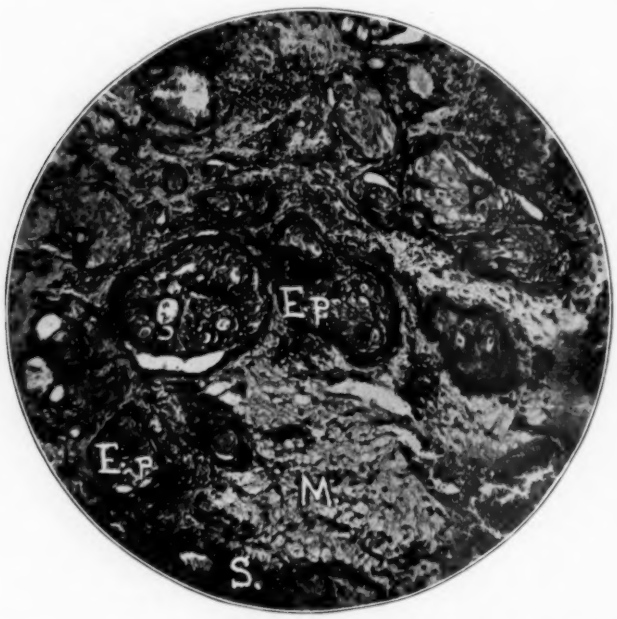

Fig. 12.—Epithelial pearls, E.p.; pearls with large eosinophil-staining cells, P; ordinary parenchymal cells, S; myxomatous tissue, M. Low power magnification.

Figure 12 is a photomicrograph of a section of the tumor demonstrating epithelial pearls. This section will explain why some pathologists regard this type of tumor as carcinomatous. Note the arrangement of the cells into typical pearls at E. p, and the unmistakable arrangement and structures of the cells at P, typical squamous cell arrangement. The cells at P stained pink, whereas the clusters of cells E. p took the alum hematoxylin stain (fig. 12).

The last section shows definite papillomatous arrangement of the parenchymal cells. Here also are seen epithelial pearls. But most interesting is the enlarged alveolus or lymph space, lined by endothelial (?) cells (A) which rest on this lining layer. Papillae (pap) project into this cavity (C). Is this the beginning of an early malignancy? (Fig. 14.)

MORPHOLOGY

As may be seen from the foregoing microscopic sections, this type of tumor possesses a peculiar morphology, not found in any tumor of any other part of the body.

In our case the tumor consisted of a mixed variety of tissues, namely, myxomatous tissue, cartilage, osteoid and fat. The parenchymatous elements of the tumor are arranged in solid masses of epithelial cells, solid strands of oval or round cells, acini or alveoli lined with oval or flattened columnar cells (the alveoli being filled with colloid or other eosinophil-staining homogenous material). Lastly, the possible malig-

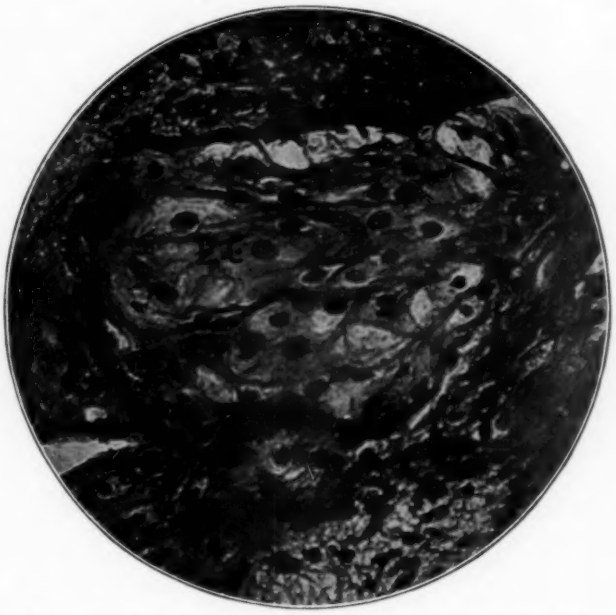

Fig. 13.—Magnification (high power) of epithelial pearl, P, in fig. 12. Large epithelial cells, E.C.

nant element of the tumor (epithelial pearls), which resemble the pearls of an epithelioma.

The literature as to the classification and general morphology of this type of complex tumor is abundant and quite confusing. Anyone sufficiently interested can get the necessary information from the excellent monograph of Francis Carter Wood,[1] Ewing's [2] textbook on Neoplastic Diseases, and from the publications of Volkmann,[3] Cohnheim,

1. Wood, Francis Carter: Ann. Surg. **39,** 1904.
2. Ewing: Neoplastic Diseases, Philadelphia, W. B. Saunders Company, 1919, pp. 714-716.
3. Volkmann: Ueber endotheliale Geschwulste, Deutsche Ztschr. f. Chir. **41,** 1895.

Hinsberg and Krompecher.[4] The endothelial, mesoblastic, and epithelial cell origin of these tumors all have their respective adherents. Cohnheim propounds that the germs of the cartilagenous tumors of the parotid are derived from the unused cartilage of the branchial arches. Some of these writers maintain that "embryonic rests" are responsible for the complex structure of these tumors.

From these theories it is not surprising to obtain the following array of a formidable nomenclature. Myxochondrolymphangio-endothelioma, endothelial sarcoma, endothelial carcinoma, myxochondro carcinoma, adenocystic epithelioma, etc.

Fig. 14.—Papillomatous arrangement of the parenchymal cells projecting into large endothelial-like cavities, pap; epithelial pearls, E.p.; eosinophil pearls, P; epithelial cells, A. Low power magnification.

COMMENT

We are inclined to the view of Wood that this class of tumor arises from misplaced embryonic mesoblast and epiblast.

Developmentally, the parotid gland arises from a furrow in the floor of the mouth in the region of the alveolobuccal groove around the angle of the mouth. Gradually this furrow of epithelium becomes pinched off and lies as a blind tube beneath the epithelium of the mouth, with its opening into the mouth cavity. Mesenchymal tissue forces its way

4. Krompecher: Ziegler Beitrage zur path. Anat. **28**:37-44.

between the tube and the alveolobuccal epithelium. The tube increasing its length pushes its way back over the masseter muscle to the neighborhood of the external ear. At this stage, solid outgrowths of cells from the wall of the duct begin to sprout. These increase in number and size and become surrounded by a mesenchymal capsule, the gland assumes the position and form of the adult structure (Hammar [5]).

In embryos from the ninth to the twelfth week, a sprout arises from the parotid duct just where it crosses the anterior margin of the masseter muscle, and, passing deeply, comes into relation with the internal pterygoid where it ends in a blind sac (Chievitz [6]).

Fig. 15.—Dissection of the normal salivary glands with their openings into the oral cavity. Anatomy: (Rauber-Kopsch). A, parotid gland; b, submaxillary gland; c, sublingual gland. A curved line joining a, b and c forms the crescent referred to in text.

We believe that the tumor under consideration is the result of one of these aberrant outgrowths having persisted, and for some unknown reason having taken on growth.

This theory will not only explain the position of our own particular tumor, which is rather rare, but also the various situations of the comparatively common mixed tumors of the parotid, situated on the outside of the face and neck. It will be remembered that in shelling out this tumor, the posterior pole was attached by a pedicle which seemed to wind around the posterior border of the jaw and the masseter muscle. Chievitz's embryonic sprout runs from before backward. The tumor

5. Hammar: Anat. Anz. **19,** 1901.

6. Chievitz, J. H.: Arch. f. Anat. u. Physiol., 1885.

in this patient appeared to have wound about from the posterior border of the parotid, under the internal pterygoid, from behind forward.

For the purpose of classification, it is much simpler to explain the many peculiarities of the cells that enter to form these tumors from the point of view of embryonic mesoblastic and epiblastic cells, with their known potentialities of becoming every one of the cells in question, than to explain away everything by the magic word "metaplasia."

The loose, spindle-celled connective tissue, cartilage, osteoid and fat of the stroma can easily be explained by the before-mentioned embryonic sprout of mesoblast. The epithelial nature of the parenchyma—solid strands of cells, alveoli, epithelial pearls—can be explained by the epithelial origin of the displaced aberrant bud of the early embryonic parotid gland.

Mixed salivary tumors are not rare. Various authors report twenty-five, twenty-seven, thirty-six, fifty-nine and sixty cases in their respective series. Most of these, however, are situated on the outside of the face and neck. They may arise from any one of the salivary glands in the oral cavity, parotid, submaxillary, sublingual, or the hundreds of discrete small glands situated underneath the mucous membrane of the mouth, labial, buccal, molar, etc. (fig. 15). It is worthy of note that the three principal salivary glands are arranged in an almost continuous crescent (fig. 15).

Wood, in his series of fifty-nine cases, reported only four as arising from the pharynx or palate. This type of tumor appears ten times more frequently in the parotid than in the submaxillary glands.

The clinical course of these tumors is that they remain small and quiescent for a long period, from ten to twelve years. Then, for some unknown reason, they begin to grow rapidly. They occur at all ages, from 1 year to 73 years.

Keen [7] reports a case in a man, aged 65, in whom the tumor lasted for twenty-four years; when finally removed it was 12 by 9 inches in size, and weighed 4¾ pounds (2,440 Gm.).

Rennie [8] removed a huge parotid tumor from an Indian woman, aged 42, who had it for twenty-eight years. It hung down from her face and neck over her chest. When removed it measured 1 foot, 9 inches by 1 foot, 7 inches, and weighed 12 pounds (5.4 Kg.).

After operation, encapsulated tumors do not usually recur. But some of them, after a period varying from two to nine years, show recurrence. In Wood's series of fifty-nine cases, 55 per cent were permanently cured, while 45 per cent showed recurrences. In recurrent

7. Keen, W. W.: Enormous Mixed Tumor of the Parotid Region, J. A. M. A. **42**:1138 (April 30) 1904.

8. Rennie: Brit. M. J. **2** (Oct. 16) 1886.

cases, the structure may remain constant, but more often it becomes increasingly cellular and malignant. But the recurrences are usually local and never involve the lymph nodes. Billroth reports a case in which nine operations were necessary during twenty-three years to remove local recurrences.

CONCLUSIONS

1. Mixed salivary tumors of the parotid may migrate to the inner side of the lower jaw and come to lie inside the oral cavity.

2. This migration may be explained by the persistence of aberrant embryonic sprouts from the primitive parotid tube.

3. The complex nature and the many peculiarities of the tissues which enter to form this type of tumor may be explained by its embryonic origin from mesoblast and epiblast.

4. From a pathologic point of view, our case is interesting and rather rare because of the many types of tissues it contained. The majority of mixed salivary tumors contain only a few types of cells.

5. Clinically, our case demonstrates the advisability of checking up by microscopic examination the roentgen-ray findings of tumors which appear to invade bone, and vice versa. A grave surgical error would have been committed if we had trusted our first roentgenogram.

6. Although this type of tumor is of slow growth, does not invade regional lymph nodes, and does not metastasize at a distance, we think it advisable to follow up even total removal of the tumor by the application of the gamma rays of radium to prevent the 45 per cent possibility of local recurrence.

THE MEDICOLEGAL IMPORTANCE OF MICROSCOPIC INVESTIGATION OF THE TEMPORAL BONE*

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ROCHESTER, N. Y.

INTRODUCTION

Attention has recently been directed to a practical importance of microscopic investigation of the temporal bone which, I believe, has not been generally recognized, namely, the medicolegal. A review of the literature [1] discloses no striking reference to it. Nevertheless, changes in a structure so intimately connected with the meninges, containing the end-organs of processes so essential to safety in everyday life, such as hearing and equilibrium, may obviously have far-reaching legal significance. Courts of law, we know, are frequently confronted with the problem of deciding negligence in fatal accident cases. Furthermore, the bearing of an accident on death months later is often of the utmost importance in the settlement of insurance claims. That a careful study of the temporal bones from such patients may prove of value is illustrated by the following cases.

REPORT OF CASES

CASE 1.[2]—*History.*—J. F., aged 54, a gardener, on the night of Jan. 15, 1924, was struck by a taxicab and died the next day. Evidence showed that L. had been driving at a moderate rate of speed and, on seeing F. in the middle of the road, had sounded his horn when some distance away; further, that F. had appeared confused and had run directly into the path of the oncoming car. There was no evidence that the deceased had been intoxicated. His son testified that F. had been hard of hearing in the right ear for several years, and that it was necessary to raise the voice when speaking with him. The defense pleaded that whether from failure to hear the approaching car, or from confusion from suddenly seeing it so near, F., by his actions, had made the accident unavoidable.

General Pathologic Examination (Pathological Institute, University of Zürich; director: late Professor Hedinger).—Multiple rib fractures; scalp laceration in the left parietal region (without evidence of fracture of the skull); laceration of the parietal and visceral pleura with hemopneumothorax and atelectasis of the left lung; laceration of the left kidney, spleen and mesentery were found. Death was considered as caused by the pneumothorax and shock.

The court acquitted the defendant on the grounds that the accident had been unavoidable. It based its opinion more on the evidence that the deceased had acted confusedly or stupidly under the circumstances than on the testimony concerning his deafness—the actual rôle of which appeared difficult to judge.

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* From the Laboratory of the Otolaryngological Polyclinic of the University of Zürich; director: Prof. Dr. F. R. Nager.

1. Even so comprehensive a book as Imhofer's "Gerichtliche Ohrenheilkunde" includes only the technic of decalcification without mention of its possible medicolegal applications. Imhofer, R.: Gerichtliche Ohrenheilkunde, Leipzig, Kabitzsch, 1920, p. 62.

2. Procedure No. 194, Archiv des Obergerichtes des Kantons Zürich, F. vs. L.

To settle the question of deafness, the temporal bones were sent to the laboratory of the Otolaryngological Institute (University of Zürich; director: Prof. Dr. Nager) for examination. After fixation, decalcification and embedding, the right temporal bone was sectioned serially in the horizontal plane, and the left in the vertical plane. Sections were stained with hemotoxylin and eosin, with the exception of every fifth section which was stained by the Van Gieson method.

Right temporal bone (horizontal sections): The external auditory canal, drum and middle ear were normal.

The upper margin of the oval window and annular ligament was normal. The lower margin, however, was surrounded by typical, spongy otosclerotic bone which involved the lower portion of the footplate of the stapes, producing a partial ankylosis, and, in addition, formed a large exostosis jutting out into the tympanic cavity. The anterior crus of the stapes was adherent to this exostosis. The main portion of the otosclerotic focus was in the lower half of the promontory below the oval window. Here the labyrinth capsule had been entirely replaced by otosclerotic bone, extending from the middle ear surface to the endosteum. A tongue-like process extended upward behind the oval window, but the annular ligament adjacent to this area was not involved. A broader process extended downward encircling the round window and produced marked exostoses into the niche as well as endostoses into the scala tympani of the outer basal coil. The round window niche, in addition to being reduced to a slit-like cavity by these exostoses, was completely filled with connective tissue, so that virtually complete closure of the niche was effected. The mucous membrane of the tympanic cavity had further sealed off the niche by growing across its mouth. The membrana tympani secundaria was ballooned outward.

Closer study of the otosclerotic area showed it to be sharply separated from the adjacent normal bone of the labyrinth capsule. The process, from a microscopic point of view, appeared to be quite active, consisting of narrow strands of bone (with no suggestion of lamellar formation) enclosing fairly large marrow spaces containing many cells and blood vessels, and showing affinity for the hemotoxylin dye. Many giant cells (osteoclasts) could be seen. In two places (namely, in the lower part of the promontory near the lateral attachment of the membrana tympani secundaria, and just below the medial margin of the oval window) the process seemed less active, consisting of thicker strands of bone, smaller marrow spaces, and fewer osteocytes and blood vessels. Both of these areas stained slightly red with eosin. The medial "Knorpelfuge" could be well seen and was not involved in the otosclerotic process.

The inner ear was, for the most part, unchanged. The macula sacculi was well preserved and normal. The only changes noted in the cochlea were in the outer turn of the basal coil, in which endostoses from the otosclerotic process projected into the scala tympani. The endosteum in this neighborhood was thickened and irregular. The mouth of the aqueductus cochleae was filled with connective tissue. The ductus endolymphaticus and *semicircular* canals were normal. There was considerable fresh blood around the eighth nerve, which otherwise was entirely normal.

Left temporal bone (vertical sections): This bone was normal. Careful study of serial sections showed no focus of otosclerosis on this side.

Summary.—There was a large focus of otosclerosis in the lower portion of the promontory of the right temporal bone, producing partial ankylosis of the stapes and closure of the round window niche through formation of exostoses.

The left temporal bone was normal.

Comment.—From the medicolegal point of view, these findings are of importance because they prove beyond question that F. was markedly deaf in his right ear. While in the opinion of the court the accident was caused

more by F.'s confusion than by deafness—the rôle of which was difficult to judge accurately from the evidence—nevertheless, the actual demonstration of pathologic changes, compatible only with marked impairment of hearing, offers strong additional support to the testimony of the defendant that he had given ample warning of his approach.

From the pathologic point of view, this case contains several points of interest. In the first place, as Lange,[3] Mayer,[4] Manasse,[5] and others, have pointed out, such unilateral cases of otosclerosis are uncommon. Symmetrical involvement of both labyrinth capsules is the rule—a symmetry which, as Mayer states, holds not only for location, but also for form, size and frequently for histologic findings as well. In Mayer's series of twelve carefully studied cases, a unilateral focus was discovered in only one case. This emphasizes the fact that a diagnosis of otosclerosis is not refuted by the finding of typical signs limited to one side.

Of further interest is the fact that, in spite of the testimony that F. had been deaf in his right ear for many years, we find in the otosclerotic focus the microscopic picture which we are accustomed to associate with activity. This agrees with the observation of Mayer,[6] that the degree of activity seems in no way dependent on the duration of the process. He mentions the case of a patient, authentically known to have been deaf for forty years, in whose labyrinth capsules apparently active foci were discovered, and further states that microscopically inactive processes may be found even in very young children.

The exostoses seen in this case illustrate a well known characteristic of otosclerosis. While they are fairly common in the region of the round window, their occurrence in the oval window is, according to Mayer,[7] relatively infrequent.

CASE 2.[8]—*History.*—J. L. aged 62, a shoemaker, on the morning of June 2, 1924, was struck by an electric automobile and carried along under the car for several feet. He died the following day without regaining consciousness. Testimony showed that the defendant had been driving at a moderate rate of speed and had given warning when some distance away from L.; further, that the deceased had appeared to start back to the sidewalk, when suddenly he turned and darted directly in front of the oncoming car, thus making the accident unavoidable. Testimony was also produced to show that L. had been hard of hearing in both ears for many years.

General Pathologic Report (Pathological Department, University of Zürich; director: late Professor Hedinger).—The subject was a rather short man (162

3. Lange, W., in Handbuch der speziellen pathologischen Anatomie und Histologie, **12**:435, 1926.

4. Mayer, O.: Untersuchungen über Otoskletrose, Vienna and Leipzig, Alfred Hölder, 1917, p. 237.

5. Manasse, P.: Handbuch der Pathologischen Anatomie des Menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1917, p. 214.

6. Mayer, O.: Untersuchungen über die Otosklerose, p. 198.

7. Mayer, O.: Untersuchungen über die Otosklerose, p. 221.

8. Procedure no. 621, Archiv des Obergerichtes des Kantons Zürich.

cm.). Struma nodosa colloides; multiple soft tissue wounds and fractures; fat emboli in the lungs and the kidneys; blood in the left pleural cavity; chronic myocarditis and endocarditis (aortic), and prostatic hypertrophy were noted. Death was considered caused by fat emboli to the lungs from the numerous fractures and, therefore, was directly due to the accident.

Although the court did not feel that the defendant had been entirely without blame, it nevertheless considered that the actions of the deceased had made the accident itself unavoidable. Without more accurate knowledge of the state of hearing, it did not feel justified in drawing any conclusions concerning the possible influence of L.'s deafness. The case, therefore, was dismissed.

The temporal bones were sent to the laboratory of the otolaryngological clinic to settle, if possible, the question of deafness.

Right temporal bone (horizontal serial sections): The external auditory canal was filled with cholesteatomatous débris. The drum was thickened and contained a large posterior marginal perforation. Through this, squamous epithelium from the outer surface of the drum and the posterior meatus wall had invaded the middle ear, forming a large cholesteatoma which filled the space behind the malleus and incus and extended upward into the epitympanum. The adjacent surface of the incus showed well marked areas of erosion. The mucous membrane of the middle ear, antrum and adjacent mastoid cells was greatly thickened and contained many small cysts.

The promontory was somewhat thickened and the window niches appeared narrow. The round window niche was partly filled with fatty and myxomatous tissue. The endosteal layer of the semicircular canals was a little irregular. The membranous labyrinth and eighth nerve were entirely normal.

Left temporal bone (vertical sections): The external auditory canal was normal. The drum was greatly thickened, due to the formation on its inner surface of a thick plate of lamellar bone. Its lower portion contained a thin scar. The mucous membrane of the middle ear was thickened, but there was no evidence of active infection. The promontory was greatly thickened by the formation of diffuse hyperostoses which had, in addition, invaded the niche of the oval window, completely surrounding the stapes, and thereby closed off the oval window. There were bony adhesions between the lateral crus of the stapes and the facial canal, as well as between the bony plate on the drum and the hyperostoses of the promontory. The niche of the round window appeared narrow and contained fatty and myxomatous tissue. There were small endostoses into the lumen of the canals.

The membranous labyrinth and eighth nerve were normal.

Summary.—These microscopic findings confirmed the testimony that L. was deaf in both ears. On the right side deafness was caused by a large cholesteatoma which completely blocked the windows; on the left side, apparently it was caused by an old chronic infection which had resulted in an unusually extensive ossifying process involving the drum and promontory, and effectively closing off the oval window. The narrowing of the window niches, together with the endostoses into the canals, were suggestive of slight endemic changes but were not sufficiently marked to have impaired the hearing. They were of interest, however, in connection with the other stigmas of endemic degeneration, namely, the short stature and the struma.

CASE 3.[9]—*History.*—A. G., aged 42, a stone-mason, on June 18, 1921, fell down a stairway, striking on his head. Although he experienced immediate vertigo, he did not lose consciousness. He was put to bed, and for the next two days suffered with intense dizziness, accompanied by nausea and periods

9. Nager, F. R.: Isolierte Basisfraktur, Schweiz. med. Wchnschr. **53**:1184, 1923. Ulrich, K.: Verletzungen des Gehörorgans bei Schädelbasisfrakturen, Acta Otolaryngol. Supplementum 6, 1926.

of vomiting. He soon observed that he had become very deaf in the left ear. Fourteen days later he was examined by Dr. Ulrich, who found complete deafness in the left ear, associated with loss of response to rotary and caloric tests. Examination of the right ear showed that it was normal except for a slight labyrinthine type of deafness. A spontaneous nystagmus of the first degree was noted on looking to the extreme right. Four weeks later, although the dizziness had not completely subsided, G. returned to work. In September, he was examined by Professor Nager, whose observations agreed with those of the previous examiner except for the note that spontaneous nystagmus had disappeared. His impression was that G. had suffered a fracture of the base of the skull involving the left labyrinth, and resulting in complete destruction of the cochlear and vestibular functions; or, less likely, that a tearing of the trunk of the eighth nerve had taken place. On the basis of this report, the insurance company awarded G. an indemnity for the disability resulting from his accident.

On Jan. 14, 1922, 215 days after the accident, G. was seized with headache and nausea while at work. On the same evening he became unconscious and rapidly developed obvious signs of meningitis. He died three days later.

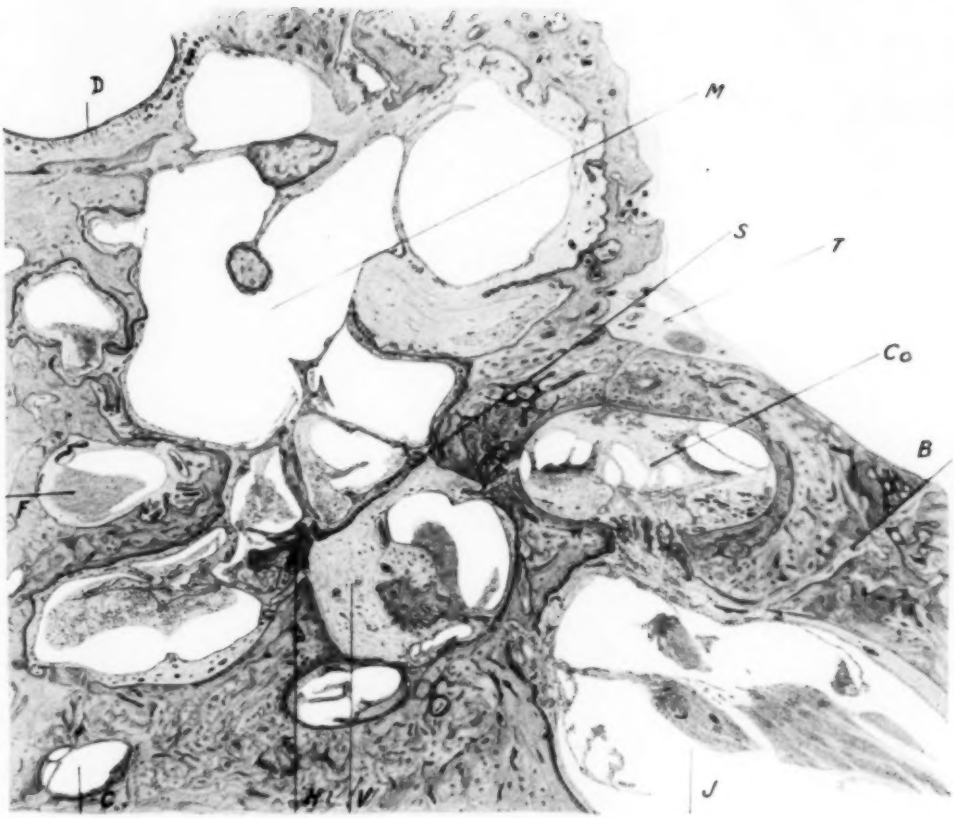
Necropsy was performed in the Pathological Institute of the University of Zürich (Professor Busse, director). A diffuse purulent meningitis was found but careful examination of the base of the skull (with particular attention to the petrous pyramid) showed no evidence of fracture. A slight mitral endocarditis was discovered and, in the absence of evidence of fracture, this was reported as the source of the terminal meningitis. It was specifically stated that the accident, which occurred seven months previously, had been without influence on the death.

G.'s family, therefore, received no compensation, because the terms of his policy insured him only against injury or death from accident.

Fortunately, the temporal bones were sent to the Laboratory of the Otolaryngological Polyclinic for study. Careful examination showed no macroscopic evidence of fracture. Microscopic examination of the left temporal bone (horizontal Zürich (Professor Busse, director). A diffuse purulent meningitis was found, serial sections) showed: The external auditory canal was normal. The drum was intact and unscarred. The mucous membrane of the tympanic cavity, as well as of the antrum and adjacent mastoid cells, was swollen, hyperemic and infiltrated with round cells. There were many clumps of pus cells. The interior of the petrous pyramid was shattered by twenty-six fractures which entered all of the labyrinth spaces, and in many places established connections with the tympanic cavity. These lines were filled only with loose connective tissue except in the periosteal (outer) layer of the labyrinth capsule in which healing by newly formed bone had taken place. Along many of these channels, pus could be seen passing from the middle ear to the labyrinth spaces. The membranous labyrinth, for the most part, was destroyed and its place was taken by connective tissue and new bone, but there remained open spaces (especially in the vestibule and scala tympani of the basal coil) which were filled with pus. The aquaeductus cochlea, as well as the ductus and saccus endolymphaticus, contained pus. The nerve endings in the cochlea and vestibule were destroyed, and pus cells could be seen passing inward along their canals. There were fractures through the areae cribrosae. The seventh and eighth nerves were surrounded by pus.

Summary.—Multiple fractures of the labyrinth capsule; chronic ossifying panlabyrinthitis; acute purulent otitis media, and otogenic purulent leptomeningitis were seen.

Conclusions.—Microscopic examination, therefore, confirmed the clinical impression that the accident had resulted in a fracture involving the capsule of the labyrinth. In fact, twenty-six fracture lines could be noted. The bleeding into the labyrinth spaces incident to the trauma was, of course, responsible for the immediate vertigo, which continued until the membranous labyrinth had been completely destroyed. This further explains the complete loss of cochlear and vestibular function noted in the examination on the fourteenth day following the accident. Later, the usual organization of the blood took place, with the formation



Case 3 (A. G.).—Horizontal section: D, drumhead; M, middle ear with thickened, hyperemic mucous membrane and clumps of pus cells; F, facial nerve. Fracture lines pass through the footplate of the stapes (S) and the posterior margin (H) of the oval window into the vestibule (V), which contains connective tissue, newly formed bone and pus cells. The internal auditory canal (J) shows clumps of pus cells entering from the vestibule along fractures through the areae cribrosea. A large, fairly well healed fracture (B) involves the anterior wall of the canal. Incompletely healed fractures enter the cochlea (Co) from the vestibule and the canal of the tensor tympani (T). The spaces of the cochlea are filled with connective tissue, new bone and clumps of pus cells. A poorly healed fracture connects the crus communis (c) with the middle ear. The tympanogenic origin of the meningitis is clearly shown.

This color plate is reproduced from Acta. Otolaryngol., Supp. 6, 1926, through the courtesy of Ulrich.

of granulation tissue and finally new bone. The fractures in this case were unusual, in that they were sharply limited to the petrous pyramid and did not involve the adjacent structures of the base of the skull. The fact that the fractures were not discovered at necropsy was readily explained by the microscopic finding that they were largely confined to the inner part of the pyramid, and, further, that those fractures involving the outer (periosteal) portion of the labyrinth capsule were completely healed with new bone.

Microscopic examination further demonstrated beyond question the bearing of the accident on death seven months later, for it showed the propagation of the acute middle ear infection along the fracture lines to the labyrinth spaces and meninges. Extension to the meninges was favored by fracture lines directly through the areae cribrosae, but it also took place along the nerve channels from the cochlea and vestibule, as well as along the aquaeductus cochleae to the subarachnoid spaces of the posterior fossa.

Therefore, microscopic examination altered the entire aspect of the case by establishing that the terminal meningitis was not metastatic from an acute endocarditis, but was caused by a direct extension of an acute otitis media to the meninges along fracture channels resulting from the accident.

On the strength of the microscopic evidence, the insurance company paid the full premium to G.'s family without delay.

Comment.—This case illustrates the importance of thorough microscopic study of the temporal bones from patients in whom fracture of the skull at one time has seemed likely. That the petrous pyramid and its surroundings are peculiarly liable to involvement in fractures of this type is well known. Furthermore, it cannot be emphasized too strongly that, as in this case, fractures involving the labyrinth capsule may exist without macroscopic evidence—due either to complete healing of their outer (periosteal) portions by new bone or, more rarely, to fractures actually limited to the interior of the pyramid. Lange,[10] Voss and Uffenorde [11] have called attention to these cases. Such fractures may create communications between the tympanic cavity and labyrinth spaces which persist incompletely healed for years, and render the patient constantly liable to a tympanogenic meningitis, the true source of which may be entirely unsuspected. Ulrich [12] states that one sixth of the patients with fractures involving the labyrinth capsule develop meningitis (immediate or delayed). The importance of thorough microscopic study of the temporal bones from such patients cannot be emphasized too strongly.

CONCLUSION

Too little attention, I feel, has been directed to otopathologic investigation in the United States. This is in marked contrast to the situation in Europe, where it is the rule to find well organized laboratories in connection with important aural clinics. This failure to develop otopathology in the United States seems largely due to the difficulties

10. Lange, W.: In Handbuch der spezrellen pathologischen Anatomie und Histologie, p. 602.

11. Voss and Uffenorde, quoted by Lange, W., in Handbuch der spezrellen pathologischen Anatomie und Histologie.

12. Ulrich, K.: Procedure no. 621, Arch. des Obergerichtes des Kantons Zürich, p. 140.

attending the preparation of temporal bones for microscopic study, the special training necessary for the interpretation of this material, as well as the popular belief that the subject has only academic interest. Three cases are reported to show that otopathology may have a practical, medicolegal value. It is true that in only one of these cases were the actual circumstances such as to make the results of the microscopic investigation of marked importance. Nevertheless, they suggest groups of cases in which the knowledge of cochlear and vestibular functions, obtainable from such examinations, will be helpful. It is hoped that the example set by a few of our clinics may be more widely followed, so that many laboratories will be available for the study not only of the scientific but also of the practical problems presented by otopathology.

OTOLOGIC COMPLICATIONS OF BASAL SKULL FRACTURE

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The otologist is especially concerned with fracture of the base of the skull because it involves the temporal bone in over one third of the cases. Within the temporal bone are contained anatomic structures of great otologic importance, such as the internal ear with its two organs of special sense for hearing and maintenance of equilibrium; the middle ear with the ossicles, mastoid cells and eustachian tube; the external ear and the membrana tympani; the seventh and eighth cranial nerves and the venous sinuses.

The otologic complications occur in 64 per cent of the cases of basal skull fractures.[1] They are of vital importance, as they may endanger the life or the future well being of the patient. The immediate result of an injury to the internal ear may be purulent labyrinthitis, meningitis and death; the remote result, partial deafness accompanied with marked tinnitus, or with disturbances of equilibrium or a total unilateral or bilateral deafness.

PATHOLOGY

The mode of injury in 380 cases of traumatic fracture of the cranial bones was as follows: falls, 219; street car, 62; assault, 51, and automobiles, 48.[2] Among 454 cases of fracture of the cranial bones, 89 per cent of which were basal, 178 involved the posterior and 166 the middle fossa.[2] Of 470 cases of skull injuries, 275, or 57 per cent, were basal skull fractures.[3]

The pars petrosa of the temporal bone is involved in 35 per cent of the basal skull fractures.[1] The excavations and canals, such as the jugular fossa, carotid canal, tympanic cavity, vestibule and the internal auditory meatus, serve to weaken it and thus predispose it to fracture.

The labyrinth is involved in 25 per cent of the basal skull fractures.[1] It is less common for the bony capsule of the labyrinth to be fractured because it is composed of dense bone, like ivory. The pars petrosa fractures most frequently in the longitudinal direction, that is, parallel to its long axis, in the weakest portion, from the jugular foramen to the

1. Bezold and Siebenmann: Textbook of Otology, Colegrove Co., 1908.
2. Le Count, E. R., and Apfelbach, C. W.: Pathologic Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries, J. A. M. A. **74**:501 (Feb. 21) 1920.
3. Brun: Der Schadelverletzte und seine Schicksale, Beitr. z. klin. Chir. **38**:192, 1903.

tegmen tympani et antri and the superior portion of the external auditory meatus. Less frequently, the fracture involves the internal auditory meatus, vestibule, cochlea and the inner wall of the middle ear. It may be fractured transversely through its base, at the tegmen and external auditory meatus or toward its apex.

Without the existence of a fracture of the pars petrosa, a subdural hemorrhage from a skull fracture may pass along the perineural lymph spaces of the auditory nerve and the aquaeductus cochlea to the labyrinth. Hemorrhage of the labyrinth similar to those occurring in fracture of the pars petrosa may occur in conditions unassociated with skull fracture, such as labyrinthine concussion from external violence or explosion. The damage to the labyrinth in basal skull fracture is usually caused by hemorrhage into the membranous labyrinth, rather than by fracture through the bony labyrinth.

Many observers have found labyrinthine hemorrhage without fracture of the bony capsule of the labyrinth in basal skull fractures. In Barnick's case,[4] blood was present in the external meatus and tympanum, and necropsy showed that the dura mater, sinus, internal ear and tympanum were not implicated by the injury. The hematotympanum was caused by a fracture through the tegmen antri. Many small hemorrhages were in the auditory and facial nerves. There were hemorrhages in the scala tympani of the basal coil of the cochlea, in the branches of the vestibular nerve and in the maculae acousticae. In Lange's [5] case, the line of fracture was parallel to the superior border and along the anterior surface of the pars petrosa. There were multiple fractures of the external auditory meatus, dislocation of the malleus and incus, rupture of the membrana tympani and hematotympanum. The labyrinthine capsule was intact, the perilymph and endolymph spaces were free from blood, the auditory nerve in the internal auditory meatus was lacerated while the facial nerve was uninjured.

Yoshii,[6] from his experimental investigations on animals, concluded that the damage to the internal ear was molecular in character, and that severe trauma caused a destruction of the acoustic end-organs, which is accounted for by the fact that the stapes is suddenly forced into the labyrinthine fluid, causing a greatly increased labyrinthine pressure.

Clinically, in some of the cases of concussion of the labyrinth in which the injury is slight, we find a temporary functional disturbance which is subsequently restored to normal. These cases are often

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4. Barnick: Ueber Bruche des Schadelgrundes und die durch bedingten Blutungen im Ohrlabyrinth, Arch. f. Ohrenh. **43**:23, 1897.

5. Lange: Anatomischer Befund an Gehororgan nach Basisfraktur, Ztschr. f. Ohrenh. **53**:37.

6. Yoshii: Experimentelle Untersuchungen über die traumatischer Schadigungen des Gehororgans, Internat. Zentralbl. f. Ohrenh. **9.**

referred to as commotio labyrinthi, and have been compared with commotio retinae—a transient edema of the retina due to trauma.

The otologic complications of basal skull fracture may be divided into the nonsuppurative and the suppurative. In the nonsuppurative group we have to consider (*a*) injury to the bone, such as fracture of the external auditory meatus, mastoid and pars petrosa with its bony labyrinth and cochlea, fracture or dislocation of the ossicles, and diploetic hemorrhage; (*b*) injury to the soft parts, such as the seventh and eighth cranial nerves, the vascular structures, membrana tympani, membranous labyrinth and Corti's organ. In the suppurative group we have to deal with the intracranial complications of otitic origin as well as with suppurative otitis media, mastoiditis and suppurative osteomyelitis of the pars petrosa.

Fracture of the bony external canal is generally associated with a fissure fracture of the superior wall of the tympanic cavity, mastoid, pars petrosa and the base of the skull. In the vast majority of cases, the membrana tympani is ruptured and there is a profuse hemorrhage from the external canal. If the membrani tympani remains intact, a hematotympanum results. Fracture of the posterior superior wall of the external auditory canal, with rupture of the posterior superior quadrant of the membrana tympani, is frequently found. A fissure or comminuted fracture of the posterior wall of the external auditory meatus may extend into the mastoid cells and antrum, resulting in an emphysema over the mastoid.

Injury of the facial nerve may occur as the result of laceration by the fractured bone, or of compression in the fallopian canal from hemorrhage. The seventh and eighth nerves are frequently injured before their entrance into the internal auditory meatus, or are involved subsequent to this. Politzer [7] reported a case of bilateral facial paralysis without disturbances of hearing. Brun [3] found facial paralysis in 17 per cent of 470 cases, while Moody [8] found it was less than 7 per cent in 547 cases of skull fracture. In 292 cases of basal skull fracture (Brun, Van Ness, Schmidt and Koehler),[3] the facial nerve was paralyzed in 45 per cent of the cases. The paralysis of the facial nerve is peripheral almost without exception.

Injury of the lateral sinus occurred in 1 per cent of Brun's cases.

If a fissure fracture extends from the external auditory meatus to involve the mastoid and infection occurs, a suppurative mastoiditis results. If the region of the sigmoid sinus is involved, a perisinus

7. Politzer: Traumatische Schadelverletzung Bluterguss aus beiden Ohren; beiderseitge Facialparalyse ohne Mehrliche Horstorung, Ztschr. f. Ohrenh. **32**:263, 1898.

8. Moody, W. B.: Traumatic Fracture of the Cranial Bones, J. A. M. A. **74**:511 (Feb. 21) 1920.

abscess, phlebitis, or thrombosis may occur. A fissure fracture involving the superior wall of the meatus externus and tegmen tympani may cause a laceration of the dura and result in pachymeningitis, extradural or brain abscess or meningitis.

In thirty-five necropsies of basal skull fracture, Borden [9] found that meningitis was present in 8 per cent of the cases. Brun,[3] in 470 cases of skull fracture, found that 8 per cent were complicated by meningitis. Moody,[8] in 547 cases of skull fracture, found that meningitis was present in 7 per cent of the cases. Basal skull fractures are more often complicated by meningitis than compound fractures of the vault. Brun's figures indicate that basal skull fractures were complicated by meningitis in 5 per cent of the cases. The chief routes of infection, in order of frequency, are (1) the nose and its accessory nasal sinuses; (2) the ear, and (3) the rhinopharynx.[3]

A suppurative labyrinthitis is usually associated with a suppurative meningitis. If a fissure fracture extends through the external and middle ear and into the internal ear, in the presence of a middle ear suppuration, there is great danger of involvement of the labyrinth and a fatal meningitis. Also, compound skull fractures may develop meningitis or brain abscess by direct continuity along the route of the involved vessels and nerves, or by metastasis.

Clinically, the meningitic complications of skull fracture may be divided into two groups, namely, early and late meningitis. The early, or fulminating, type of meningitis runs a very short course, the patient dying in from two to seven days after the accident. In the late cases of meningitis, the onset may occur in the second week to two months after the injury. Politzer [10] reported the case of a patient with basal skull fracture in whom a fatal meningitis developed seven weeks after injury. This case was unique in several respects. While walking, the patient fell, striking his head on the hard pavement. The drum membrane was found intact and he was completely deaf. Necropsy showed a bilateral fracture of the pars petrosa with fracture of each labyrinth through the vestibule and extensive hemorrhage into both labyrinths. A suppurative labyrinthitis and meningitis were present. Klestadt [11] reported a case of late meningitis after labyrinth fracture, in which the patient developed a fatal meningitis thirty weeks after the accident.

Brain abscess as a complication of skull fracture occurred in 1 per cent of Brun's cases. According to von Bergmann, acute brain abscess develops in three to five weeks, and R. Meyers states that

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9. Borden, C. R. C.: Otologic and Rhinologic Complications of Skull Fracture, J. A. M. A. **53**:429 (Aug. 7) 1909.

10. Politzer: Diseases of the Ear, Philadelphia, Lea Brothers, 1909.

11. Klestadt: Spat Meningitis nach labyrinthfraktur, Verhandl. d. deutsch. otol. Gesellsch. **22**:229, 1913.

chronic brain abscess requires six weeks to develop.[3] Brain abscess may remain symptomless for months or even years. Harrison observed a latent period of eleven years.[3]

DIAGNOSIS

The otologic aids in the diagnosis of basal skull fracture are:

1. Profuse bleeding from the ear. Moody [8] found bleeding from the ear in 24 per cent of 547 cases of fracture of the skull. Ransohoff,[12] in 190 cases of basal skull fractures, found bleeding from one ear in 31 per cent and from both ears in 4 per cent. Borden [9] found hemorrhage from one or both ears in 75 per cent of 346 cases of basal skull fracture. Hemorrhage occurred from both ears in 11 per cent.

The duration of the hemorrhage from the ear is usually short; ordinarily, it ceases on the first or second day, and it may be first noticed as a blood clot in the external canal and in the torn membrana tympani. Brun mentions a case of profuse hemorrhage from the ear in a basal skull fracture which lasted nine days and caused a marked anemia. Usually the hemorrhage stops completely, and all discharge is absent or only a slight serous secretion is left. The source of the hemorrhage may be from the bony external auditory canal, membrana tympani, mucosa of the tympanic cavity, diploetic veins of the fractured bone and the meningeal vessels. Buck [13] called attention to the occurrence of a copious and protracted hemorrhage from the tympanic artery following simple fracture of the glaserian fissure.

Visible bleeding from the ear is not a necessary accompaniment of fracture of the temporal bone. Buck [13] cites a case with necropsy findings of fracture of the pars petrosa without hemorrhage from the ear, and Politzer [10] cites the case of a bilateral fracture of the pars petrosa.

It is necessary to exclude the entrance of blood into the canal from some external source. A rupture of the membrana tympani or a fracture of the external auditory canal may be unassociated with a basal skull fracture. In a simple fracture of the bony external auditory meatus due to a blow on the face or chin, the line of fracture is in the antero-inferior portion, and extends inward to involve the anterior part of the membrana tympani.

2. The escape of cerebrospinal fluid from the ear. This is less common than bleeding from the ear. The amount of the discharge is greater than that of blood, and is of longer duration. It always signifies a basal skull fracture involving the temporal bone, and is present in 20 per cent of the basal skull fractures.[1] It is a pathognomonic sign that

12. Ransohoff, quoted by Speed: Fractures and Dislocations, Philadelphia, Lea and Febiger, 1916.

13. Buck: Fractures of the Temporal Bone, Am. J. Otol. **2**, 1880.

the subarachnoid space has been opened. This usually occurs over the tegmen or in the sheaths of the seventh and eighth cranial nerves in the internal auditory meatus. The quantity of the cerebrospinal fluid that escapes may be as high as 600 cc. in twenty-four hours.[14] It usually escapes through a laceration in the membrana tympani, or if this is intact through a fissure in the bony external meatus. Blood and cerebrospinal fluid may escape simultaneously.

3. The presence of brain substance in the external auditory canal. This is positive evidence of a basal skull fracture involving the temporal bone.

4. Fracture of the posterior superior wall of the bony external auditory canal with rupture of the posterior superior quadrant of the membrana tympani. Luxation or fracture of the ossicles may be present. There may be lesion of the soft parts adjacent to Schrapnell's membrane.

5. Subcutaneous emphysema over the mastoid region. This is indicative of a fracture involving the mastoid cells which communicates with the subcutaneous tissues. (I had such a case of basal skull fracture on my service at Cook County Hospital.)

6. Peripheral facial paralysis. This is located on the side of the injured nerve. If deafness is present, it is on the same side as the deafness. When the chorda tympani nerve is involved, there is loss of taste on the anterior two thirds of the tongue on the side of the facial paralysis.

7. Disturbances of the auditory apparatus. The peripheral portion of the cochlear nerve with its end-organ, Corti's organ, located in the membranous portion of the cochlea is affected in the vast majority of cases. The central portion of the cochlear nerve with its auditory tract in the midbrain and cerebrum and the auditory center in the temporal lobe is rarely involved.

There is a disturbance of hearing in 24 per cent of the cases of basal skull fracture as a result of injury to the middle and internal ear.[1] In twenty-seven cases of verified skull fracture, especially of the base, Nager [15] found seven cases of unilateral total deafness and eleven cases of defective hearing due to injury of the internal ear. Middle ear deafness was present in three cases. In six of the cases, there was defective hearing present previous to the accident, as evidenced by a history of occupational deafness or middle ear suppuration. In five cases the hearing was normal, although the line of fracture partly extended

14. Ballance: Injuries of the Middle and Internal Ear, Proc. Roy. Soc. Med., Section on Otology **4**:87-106, 1910-1911.

15. Nager: Zur Begutachtung traumatischer Ohraffektion, Cor.-Bl. f. schweiz. Aerzte **44**:486, 1914.

through the external auditory canal. The fact that Nager found the cochlear nerve was involved in 67 per cent of the cases of skull fracture contradicts the usual statement (Brun, von Bergmann) that the facial nerve is the one the most frequently involved in skull injuries, and emphasizes the importance of making the functional hearing test in these cases. Graf tested the hearing of thirty-nine subjects who had recovered from fracture of the skull, in whom it was not suspected that the pars petrosa was involved. He found that hearing was normal in only twelve cases.

Often it is impossible to make the functional hearing tests immediately following the injury on account of the coincident stupor or unconsciousness of the patient. The examination should not be attempted until the patient has recovered from shock or clouding of the sensorium, so as to be able to cooperate intelligently with the examiner.

Deafness or disturbance of hearing is present on the side of the lesion, and is usually of the nerve type of deafness in which the Rinne test is positive; the Weber test is localized to the sound side in unilateral involvement, there is lowering of the high tone limit and a slight diminution of bone conduction. In the incomplete involvement of the cochlear nerve, a severe and persistent tinnitus or paracusis may be associated. Deafness for the high tones is due to complete atrophy of the nerve fibers in the lamina spiralis and changes in the ganglion cells in the lower part of the cochlea (Passow). The cochlear nerve is the more vulnerable portion of the eighth nerve. If a unilateral or a bilateral total deafness appears gradually after the injury, it usually signifies fracture of the pars petrosa. A complete loss of hearing occurred in eight or ten cases of skull fracture (Buck).

8. Disturbances of the vestibular apparatus. Like the cochlear apparatus, the vestibular apparatus may be divided into its peripheral and central portions. The peripheral portion includes the static labyrinth and the vestibular nerve. The central portion includes the vestibulocerebellocerebral tracts and the cerebral center in the temporal lobe. Also, the peripheral and not the central portion is more frequently involved in skull fractures.

The symptoms indicating injury of the peripheral portion of the vestibular apparatus are vertigo, spontaneous nystagmus toward the healthy side, past pointing toward the diseased side, nausea, vomiting and disturbances of equilibrium. Tinnitus and deafness are usually associated. The patient prefers to lie on the healthy side because the spontaneous nystagmus is toward that side. The vertigo, of slight duration and annoying in the beginning, is toward the diseased side. It gradually disappears completely or is only noticeable in stooping. Vertigo and nystagmus usually disappear after one year. Nystagmus

was present in 25 per cent of the patients in the Breslau Sanitarium for accident cases. In 1906, Rhese found a much higher percentage.

The Bárány tests of the vestibular apparatus in the destructive type of lesion of the static labyrinth show an absence of response to the turning and caloric tests, and if the vestibular nerve is involved the galvanic test will be absent on the affected side. A severe injury to the vestibular apparatus nearly always involves the cochlear apparatus, and usually total deafness on the affected side is present.

A comparison of the relative value of the roentgenologic and otologic aids in the diagnosis of basal skull fractures would be of interest. A positive roentgenologic diagnosis of skull fracture was made in 133 instances, of which 105 were not subsequently confirmed by operation or necropsy, and there was no roentgenographic evidence of skull fracture in ninety-six cases, of which seventeen were found to have a skull fracture at necropsy.[8]

Because of the great difficulty of detecting a fissure fracture of the skull, and especially of the base roentgenologically in suitable cases, we should add the functional tests of the cochlear and vestibular nerves to our diagnostic armamentarium.

PROGNOSIS

The mortality in 470 skull fractures was 35 per cent, and 70 per cent of the fatal cases had involvement of the base (Brun). The mortality was 81 per cent in 547 cases (Moody). During the past five years, the mortality at the Cook County Hospital was 36 per cent;[18] Murney[3] placed the mortality of fractures of the vault at 46 per cent, and fractures of the base at 69 per cent. Phelps[16] found a 40 per cent mortality in 285 cases of basal skull fracture with hemorrhage from the ear. Blahd[17] reports a 33 per cent mortality in fifty-nine skull fractures, of which the mortality of vault fracture was but 13 per cent, while in fracture of the base it was 65 per cent.

Von Bergmann's rule was that patients with skull fractures who survive over forty-eight hours gave a favorable prognosis. At the Cook County Hospital, 25 per cent of the patients with admitted skull fractures die within the first thirty-six hours.[18]

The fatal cases of skull fracture may be divided into two groups. The vast majority belong to the group that succumb immediately as the result of intracranial hemorrhage, medullary compression, brain injury and shock; the remainder, a small minority, die from infection.

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16. Phelps: One Thousand Cases of Head Injury, Ann. Surg. **49**:449.
17. Blahd, M. E.: Fracture of the Skull and Its Complications, Am. J. Surg. **37**:33 (Feb.) 1923.
18. Jackson, Harry: Personal communication.

Skull fractures were complicated by meningitis in 8 per cent and by brain abscess in 1 per cent (Brun); 5 per cent of the meningitis cases were associated with a fracture of the base of the skull; of the latter, the anterior fossa was most often involved, implicating the ethmoid and frontal sinus. After these came fracture of the temporal bone and, lastly, fracture through the roof of the sphenoid sinus. We may conclude that otitic meningitis caused by skull fracture is an infrequent occurrence, and does not comprise more than 2 to 3 per cent of the cases of skull fracture. It is with this group that the otologist is immediately concerned. The prophylaxis of infection via the ear at the time of the injury is extremely important. It is a well established fact that a basal skull fracture with a fracture into the labyrinth in the presence of a middle ear suppuration is certain to result in a fatal meningitis. This is especially so in the patients with chronic suppurative otitis media, with fetid discharge, granulations, polypi or cholesteatoma. The provision of drainage early in these cases by means of a radical mastoid operation may be the means of preventing a fatal meningitis. The incidence of this type of cases must be comparatively small. I was unable to find in the literature any reference to the percentage of cases of basal skull fracture involving the temporal bone that were associated with a chronic suppurative otitis media.

Has the discharge of blood or cerebrospinal fluid from the ear any prognostic significance? The advantage is that it lessens the increased intracranial pressure which may be the immediate cause of death. It obviates the necessity for lumbar puncture in those patients with a discharge of cerebrospinal fluid from the ear. The disadvantage is that it predisposes to infection, and to suppurative intracranial complications. The hemorrhage per se may be so severe as to cause death. The mortality of 285 basal skull fractures with hemorrhage from the ear was 40 per cent.[16]

Considered from the point of view of morbidity, the prognosis concerns the otologist with regard to the disturbances of hearing and equilibrium, and has to do with the future well being and working capacity of the individual. He may entirely recover or be partially or permanently incapacitated and unable to resume his former occupation, or, indeed, any occupation. While the patient may improve in the course of time, otology cannot offer any material help in most of these cases. Complete recovery of hearing may occur in some cases of labyrinthine concussion, but permanent and total, unilateral or bilateral, deafness usually results after fracture through the labyrinth. In the Zurich clinic,[1] it was found that both labyrinths were involved in 14 per cent of patients with basal skull fractures who recovered. Vertigo is most marked at the onset, and gradually diminishes until labyrinthine compensation has become completely established when it ceases. In

some of the cases of labyrinthine concussion with hemorrhage in the labyrinth and involving the cochlear nerve, there occurs a gradual but progressive loss of hearing associated with tinnitus, which may continue until there is complete atrophy of the cochlear nerve.

The patients in whom there is a basal skull fracture are often much better off than those with lesser injuries, because, while they may be deaf in the injured ear, they almost never complain of attacks of vertigo and tinnitus of labyrinthine origin.[19]

TREATMENT

In the consideration of the treatment of the otologic complications of basal skull fractures, I wish to emphasize the importance of the prophylactic treatment with regard to the prevention of the otitic intracranial complications, especially meningitis. Otology can only offer aid in the immediate otologic complications of basal skull fractures in the rôle of prophylaxis; in the remote complications its curative powers are practically nil.

The Treatment of Hemorrhage or Discharge of Cerebrospinal Fluid from the Ear.—This is of primary importance in the prevention of infection. Getting any fluid into the ear should be avoided; there should not be syringing or instillations. The external canal may be kept clean by mopping with sterile cotton applicators moistened with alcohol. The canal should never be packed. A voluminous sterile dressing should be applied. Care should be exercised when washing or bathing not to get water into the ear canal. These conditions are open or compound fractures, and if infection can be prevented the patients may recover. By reason of their being compound, they have been provided with an avenue of escape for the increased intracranial pressure by means of a traumatic decompression.

In case the hemorrhage from the ear is severe and threatening, its source should be investigated under the most rigid asepsis.

The Treatment of Basal Skull Fracture Involving the Temporal Bone in the Presence of Middle Ear Suppuration.—One of the very few indications for surgical interference in skull fractures is to prevent infection. In basal skull fractures accompanied by middle ear suppuration, there is an additional danger to the patient in that we have the avenue of the fracture for the dissemination of the infection contained in the middle ear, which may lead to the labyrinth with its intimate connection with the subarachnoid space, resulting in the production of an acute diffuse suppurative leptomeningitis and death.

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19. Ruttin: Die Begutachtung dauernder Folgen der Verletzungen des innern Ohres, Monatschr. f. Ohrenh. **44.**

What has otology to offer as a prophylaxis against this? In the early or fulminating type of meningitis we are helpless, but in the late or delayed cases of meningitis a fatal meningitis may be prevented by providing early prophylactic drainage of the middle ear suppuration. This applies especially to the cases of chronic suppurative otitis media with granulations, polypi or cholesteatoma, in which a radical mastoidectomy is indicated.

The indications for mastoidectomy should be broad and less exacting in the presence of a basal skull fracture than are ordinarily required. In those subjects in whom the middle ear becomes secondarily infected as the result of the basal skull fracture, or was previously infected, and in whom there is stasis of the purulent discharge from the ear due to narrowing of the external canal, when acute mastoiditis develops, where the fracture extends through the mastoid cortex and involves the mastoid cells and antrum causing emphysema over the mastoid region, or in which there is the slightest suspicion that the middle ear infection is progressing toward the internal ear to involve the labyrinth and the meninges, a mastoidectomy should be done immediately.

We realize that a prophylactic mastoidectomy, to provide for additional drainage of the middle ear suppuration, subjects the patient to operative trauma, but it is the lesser of two evils, providing that the patient is in condition to stand the necessary operative interference.

It goes without saying that we must refrain from operating in the presence of shock or on moribund patients. It is not necessary to employ general anesthesia in these cases, as the mastoidectomy can be done without pain under local anesthesia.

It is doubtless true that prophylactic mastoidectomy will not prevent the development of meningitis in all of these cases, but if performed early enough, it is the only ray of hope that otology has to offer.

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APHONIA

THE REPORT OF THREE CASES *

W. R. WATSON, M.D.

PHILADELPHIA

Aphonia is defined by Webster as being a "loss of voice or vocal utterance, due to disorder of the vocal cords." Moreover, to enlarge on this simple definition, vocal utterance is defined as an "element of speech consisting of pure vocal tone." Thus, nothwithstanding an individual by whispering may express himself in words and make himself understood from a variable distance away, he has nevertheless lost his voice and is suffering from what is termed aphonia. There are certain aphasias, however, whereof it is said the patient "is unable to speak." But no one ever thinks of calling this aphonia. Nor does vocal utterance in these particular cases depend on the cords.

Aphonia is a condition that so commonly confronts the modern laryngologist that apparently it is scarcely deemed worth while reporting. This subject was more popular in the past, however, for the older literature is full of it. But, curiously enough, I found that rarely indeed was this affection attributed to physical causes. Almost invariably, the case reports were of patients suffering from "functional aphonia."

ETIOLOGY

The study of the particular etiology in certain cases that I have attended has been well worth the time I have spent on them, and I have chosen a group of three out of a number that have appeared in the clinics of the Episcopal Hospital as offering sufficient interest to report, especially as one of them offers a rebuttal, at least tentatively, to the claim of certain laryngologists that primary tuberculosis of the larynx cannot exist.

There are several exhaustive classifications giving the causes of aphonia. I will not repeat them, but will mention a few of the more common causes, more or less in the order of their frequency so far as my experience has taught me.

1. Tuberculosis is the commonest cause; that is, of the comparatively permanent aphonias. As a matter of fact, acute catarrhal laryngitis is the most frequent cause of all, but as this condition is limited to only several days' duration, it does not come within the purview of this paper. Tuberculosis may act locally on the cord or ventricular band

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* Read before the otological section of the College of Physicians, Philadelphia, April 21, 1926.

to cause aphonia, or, by distortion of the arytenoids by means of edema or tumerous tubercles, may prevent the cord from approximating.

2. Syphilis is a common cause according to my experience. It may act locally or indirectly; indirectly by pressure of a syphilitic tumor on the cortical centers, or on the trunk of the tenth cranial nerve, or on the recurrent laryngeal, or even on the terminal endings of the latter in the larynx, though this is rare. Unusual conditions resulting from syphilis are the aphonias noted in locomotor ataxia and paresis of the cortical centers from a broken down or softening gumma.

3. Benign or malignant growths around or on the vocal cords is a fairly frequent cause, though in the majority of these cases the patients do not wholly lose their voices. They usually speak in a husky or whistling tone, which is so characteristic that the condition is often diagnosed without examination. The ordinary papilloma is the most frequent offender.

4. Paralysis of the intrinsic muscles of the larynx through neuritis of the controlling nerves, caused by a focal infection, or by pressure on the trunk of the pneumogastric or recurrent laryngeal nerves by pericardial or pleural effusions and mediastinal growths, or by aneurysm of the arch of the aorta, or even by a hypertrophied thyroid gland, as well as by enlarged glands in the neck or tumors of the esophagus.

5. Paralysis of the cords or intrinsic muscles in certain forms of infectious diseases, such as typhoid fever and diphtheria, which, while not permanent, are at times of several weeks duration. The aphonias found in the exanthematous diseases are as evanescent as those in acute catarrhal laryngitis.

6. A rare cause of aphonia would be embolism, causing an infarct in the cortex of the brain, or apoplexy in the same area, or a tumor, mild or malignant, pressing on the cortical centers governing the muscles of the larynx.

7. Shock, from concussion of the brain, as from a blow on the head, providing it causes incoordination of the centers in the cortex; or from a fractured skull lacerating the trunk of the tenth nerve, or from a lacerated wound in the neck, or an incised wound, such as would be made in a thyroidectomy, causing trauma of the motor nerves of the larynx.

Besides the organic causes for aphonia, the latter condition may be found in almost any neurosis.

The three cases which I desire to report are as follows.

REPORT OF CASES

CASE 1.—E. D., a bartender, aged 62, was admitted to the wards of the Episcopal Hospital on May 13, 1925. The family history was negative.

Several months before coming to the clinic, he was becoming hoarse for no apparent reason, and a short time later lost his voice entirely. There was no pain in the throat, either on swallowing or otherwise. He coughed seldom, and not much at any time. He had lost little weight. No fever or night sweats were present at any time. In other words, the only symptom from which he suffered was the loss of voice.

The patient appeared in the dispensary, giving the history as stated in the foregoing. Indirect laryngoscopy revealed a swelling in the posterior commissure extending up between the arytenoids, which held the cords apart and so fixed as to prevent them from approximating, although there was distinct though somewhat limited abduction on inspiration. The tumor was small, flat and rough looking; that is to say it had not the smooth and velvety appearance of mucous membrane, and no ulceration could be detected. The epiglottis was free from any defect and the arytenoids were of a normal color and not enlarged. No glandular enlargements could be found in the neck or elsewhere. The vocal cords were of a normal color, except perhaps that they were slightly pink in the part contiguous to the tumor.

A Wassermann test was made which proved negative. Although the condition had already been tentatively diagnosed as malignant, the patient was sent to the chest clinic for a careful study of his lungs and sputum. Several exhaustive examinations were made, including the usual frequent roentgen-ray examinations of his chest, but no evidence of pulmonary tuberculosis was found.

Therefore, since no subjective symptoms of tuberculosis could be found, and especially after the assurance by the chest clinic, and also on account of a vigorous denial of venereal history as well as a negative Wassermann reaction, the laryngologic section of the hospital felt fairly well justified in making their first diagnosis of malignancy definite. His age, as well as the appearance of the tumor, were factors in this conclusion. He was sent to the hospital merely to substantiate this diagnosis by biopsy.

A specimen was collected by direct laryngoscopy, and sent to the laboratory with the expectation of a diagnosis of carcinoma, but no cells simulating either benign or malignant tumor were discovered. Typical tubercles were found, however, though a search after staining the sections for acid-fast bacilli was ineffectual.

This would almost appear, therefore, to be primary tuberculosis of the larynx. I belong to the group of laryngologists who believe that there is no such thing as primary tuberculosis of the larynx. At any rate, the condition of this patient comes closer to this diagnosis than any other I have ever seen. I saw this patient frequently thereafter, the last time on September 22, and his symptoms had not changed. He felt well and had lost a little weight during the summer, but had regained nearly all of it since. He coughed no more than he did before. There had been no fever or night sweats. He had had no pain on eating and no discomfort elsewhere except aphonia. On examination of his larynx from time to time, it was observed that the lesion in his posterior commissure had extended to the ventricular band on the right side. The latter was so swollen that the cord on that side could not be seen.

CASE 2.—R. L., a negro, aged 20, a laborer, had practically the same lack of symptoms as in case 1; that is, there was no discomfort other than loss of voice. Moreover, there was no loss in weight, no elevation of temperature when he appeared in the dispensary, no night sweats, and no cough to speak of. The family history was negative.

This patient appeared in the clinic of the Episcopal Hospital, May 27, 1925, with the statement that he had begun to get hoarse two years before when a fish bone stuck in his throat. He had succeeded in coughing up the fish bone, but the hoarseness persisted. For several months he has been speaking only in a whisper.

Examination disclosed enormous tonsils and indirect laryngoscopy a practically normal larynx, with the exception of a right ventricular band so swollen as to eclipse totally the vocal cord on that side; moreover, this band apparently was bound down to the vocal cord. There was normal abduction of the left cord on inspiration, but the right cord could not be seen. There was no edematous swelling of the epiglottis or the arytenoids, nor could the color of the mucous

membrane of the larynx be called abnormal. A gland or two of the anterior chain in his neck could be felt, which, however, was attributed to the infected tonsils.

Two Wassermann tests, and even an additional provocative Wassermann test, were negative. In the meantime, in the forlorn hope that they might have some inflammatory effect on the larynx, the tonsils were removed. This had no effect on either the phonation or the swelling of the ventricular band.

This patient was also sent to the chest clinic, and while no microscopic evidence was found in the several specimens of sputum, nor were any definite signs found by physical examination, it was discovered that he invariably ran a daily temperature of 99.3 and 99.4 F., which seemed to be sufficient to justify a diagnosis of incipient pulmonary tuberculosis.

During the last days of September, 1925, we were able to locate this patient as a laborer on the subway, but could not get him to come to the clinic. He sent back word that he was feeling fine, but he was still speaking in a whisper.

CASE 3.—A. N., a white woman, aged 60, was admitted to the medical wards of the Episcopal Hospital on May 17, 1925, for study. Her only symptoms were aphonia and cough, and her family history was negative. Her health had been good except for cough and colds. Twenty years before presentation she had suffered from abscesses in both legs. She became hoarse every time she acquired a cold, which would persist for weeks. She had never expectorated blood. She perspired a good deal at night; there were no dyspnea or headaches, but she had vertigo at times. There was no pain on swallowing. She was married when she was 40, and was never pregnant.

Her present illness started with a bad cold in the chest about ten weeks prior to examination, with productive coughing. The sputum was yellowish, thick and tenacious, but there was no blood. There was no pain in the chest, no chills or fever.

Physical examination on admittance showed nothing of importance.

The patient was referred to the laryngologic service of the hospital, and on examination by mirror, a paralysis of the right vocal cord was revealed. A Wassermann test and a roentgen-ray study of her chest in the region of the right recurrent laryngeal nerve were suggested. The Wassermann reaction was 4 plus. The roentgen-ray report by Dr. Ralph Bromer was as follows: "Extreme thickening of the hilum in both lungs, extending into the apexes, is present. Nothing is found with reference to aneurysm of the aorta or mediastinal effusions or tumors. It is possible that the appearance of the lungs may be due either to hilum tuberculosis or to syphilis."

Examination of the blood and urine was uninteresting. The sputum was normal.

Neurologic study by Dr. George Wilson disclosed the following: "Bilateral involvement of the eleventh and twelfth, and unilateral involvement of the tenth cranial nerves, central in origin."

The patient was given several intravenous doses of arsphenamine, with apparently no result so far as her phonation was concerned. This patient was seen again on October 29, and it was noted that the right cord had slightly increased in function, but was still badly paralyzed. The voice was better. The whisper had become a voice, hoarse and croaking as it was. At that time the patient had received fifteen intravenous applications of arsphenamine in all.

COMMENT

As I have mentioned, this patient had been suffering from aphonia for ten weeks, and, if we except the cough, from nothing else. The appearance of the larynx was perfectly normal; there was not the slightest sign of either acute or chronic inflammation or edema, despite her cough. The only abnormality, in fact, was the paralyzed cord, which,

while not exactly in the so-called cadaveric position, stood far enough apart from the other cord so that attempted phonation was ineffectual. The question is, therefore, was she suffering from a functional aphonia at this time, or did she actually have paralysis of the cord from a central syphilitic lesion? It would almost seem that if one could forget the 4 plus Wassermann reaction and the neurologic report, as well as the patient's apparent recovery by intravenous medication, we might think of it as being functional in character.

In this respect it might be interesting to repeat the opinions of certain members of the Royal Society of Medicine, London, with reference to unilateral paralysis of the cord, given in a recent abstract of the proceedings of the latter society. The case which caused the discussion was that of a man, aged 56, with immobility of the cord for eighteen months, which was reported by M. Vlasto, F.R.C.S.

The history was as follows:

> The subject was a painter, who had been hoarse for eighteen months. The laryngeal parts were congested. The left false cord was so swollen that the true cord could not be seen. The Wassermann reaction was negative. The chest was normal, and no dysphagia was present. Shortly after the first examination some of the laryngeal swelling subsided, and then it became apparent that the left cord was fixed. The condition had not changed in six months. Opinion was invited.

In the discussion, Mr. Herbert Tilley referred to a paper he had published in 1918 in which he had followed up the after histories of twenty-three cases of paralysis of the cords in private practice during twenty years. No reply was received from ten patients; seven had completely regained the function of the cords; three had died, and three had continued in the same condition as when first seen. The fatal cases were due to aneurysm of the aorta and to malignant disease of the mediastinum and gullet. The seven recoveries were probably due to ankylosis of the crico-artenoid joint.

Sir William Milligan thought that many of these cases were functional, though this went unrecognized when only one cord was affected. He had seen several cases thought to be due to organic disease, and when they were presented several years later he found that they had regained their function without treatment, which suggested that they were almost certainly functional. The possibility of a unilaterally paralyzed cord being of functional origin should not be overlooked.

Sir St. Clair Thompson said that possibly there was functional paresis of one cord. He had watched cases, and sometimes could not make up his mind whether there was complete mobility of a cord or not. He had had eleven cases of paralysis of the cord in tuberculous patients with nothing wrong in their larynx. In five of the eleven there was recovery of movement. Of the remainder, one or two disappeared from observation, and in two or three the condition had become permanent after several years.

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COMPARISON OF THE RESULTS MADE WITH TWO TYPES OF AUDIOMETER*

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In the February issue of this journal, Bunch[1] gave a number of charts showing a comparison of the results of acuity tests of hearing taken by two different types of audiometer. These types were the 1-A audiometer, manufactured by the Western Electric Company, and the pitch range audiometer, developed at the State University of Iowa. They were called the "A" and "B" audiometers, respectively, a designation which will also be used in this paper.

As a large number of audiograms have been published by Dean and Bunch, together with the clinical history of the cases, it occurred to me that it would be of assistance to those otologists who are using sensation units in plotting audiograms to be able to redraw the audiograms of Dean and Bunch on the new scale. For this purpose, a set of calibration curves has been deduced from the results published by Dr. Bunch in the paper cited in the foregoing. This set of calibration curves also enables one to transfer from the sensation unit scale to the Dean and Bunch scale whenever that is desired.

CALIBRATION CURVES

These calibration curves were obtained by plotting at any one frequency readings obtained on the "B" audiometer as "abscissae," and those obtained on the "A" audiometer as "ordinates." Calibration curves are given in figures 1 and 2 for frequencies 60, 150, 330, 770, 1,550, 2,500, 3,130 and 4,000. Interpolations can readily be made for frequencies between those mentioned. If it is desired, calibration curves at other frequencies can be obtained in the manner before mentioned. Such calibration curves also enable one to reduce the results of Dean and Bunch to absolute units, that is, dynes pressure on the ear drum, when that is desired. The method of transferring from sensation units to dynes pressure is explained in a paper entitled "Physical Measurements of Audition and Their Bearing on the Theory of Hearing," which was published in the September, 1923, number of the *Journal of the Franklin Institute*.[2]

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* A discussion of a paper with the same title by Dr. C. C. Bunch.

1. Bunch, C. C.: Auditory Acuity Tests. Comparison of the Results Made with Two Types of Audiometers, Arch. Otolaryng. **3**:108 (Feb.) 1926.

2. More accurate calibration curves could be obtained by comparing directly the loudness of the tone from the two instruments. Noting the readings thus taken, they could be plotted in the manner before described.

By means of these calibration curves, all of the audiograms given in Dr. Bunch's paper are redrawn on the same scale so as to make comparisons more easily. These are shown in the charts given at the end of this paper. They bear the figure numbers used by Dr. Bunch, but to avoid confusion with the figures referred to in this discussion, they will be referred to as charts.

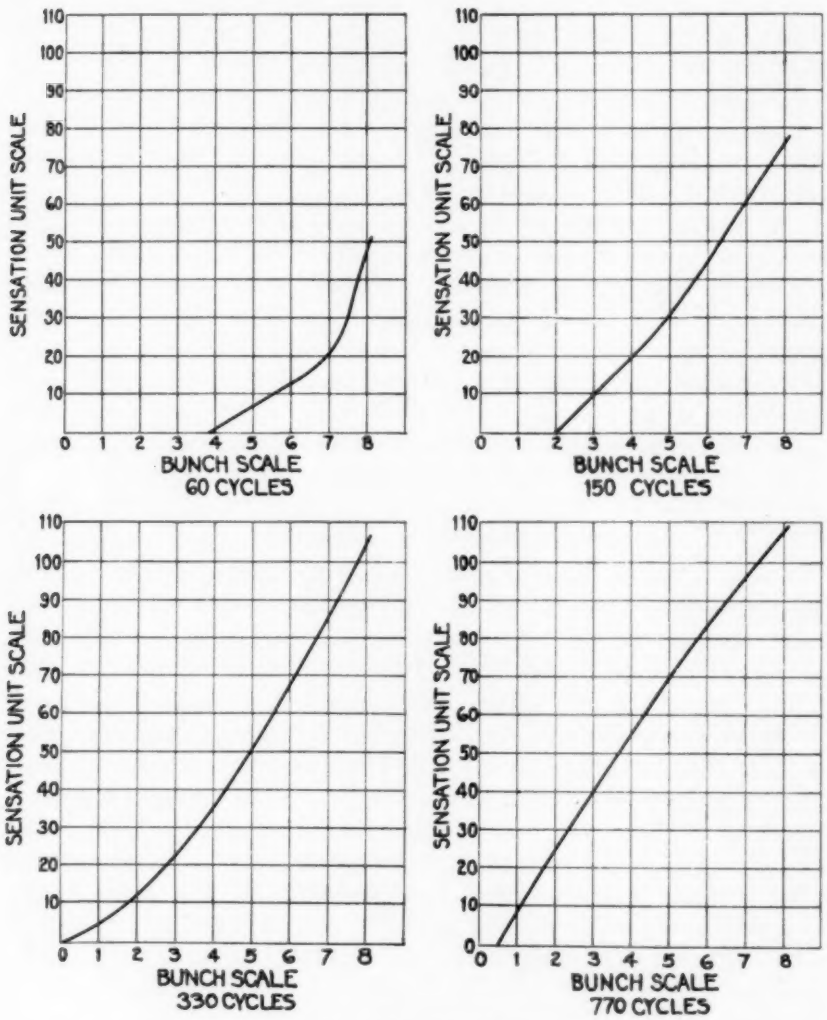


Fig. 1.—Calibration of Dean and Bunch auditometer in sensation units.

The main differences in the two sets of curves cannot be due to the type of instrument or the method of making the test. It must be due to variations in the constancy of one or both audiometers, or to variations in the judgments of the patient being tested or to both of these causes.

The agreement is fairly good except in cases of extreme deafness or for high frequencies when the differences seem larger than one would expect. However, it must be admitted that the judgment of a patient who is very hard of hearing is sometimes open to question.

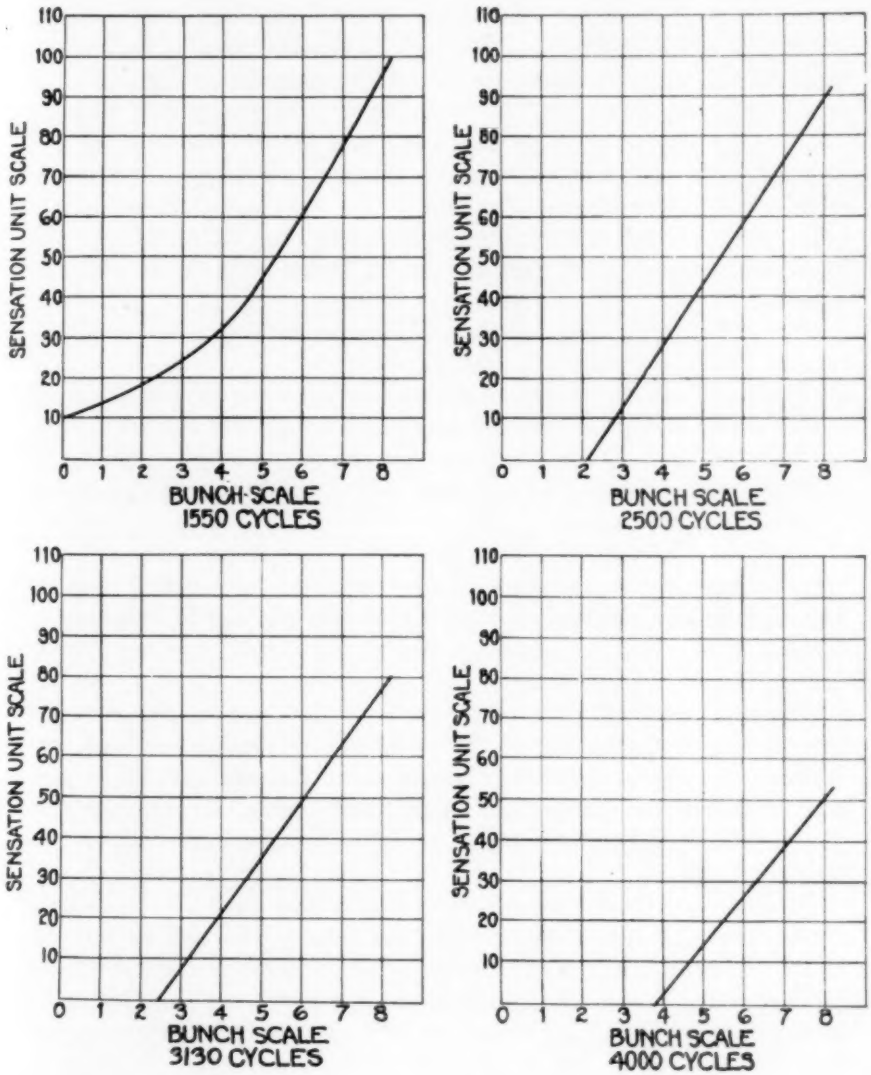


Fig. 2.—Calibration of Dean and Bunch audiometer in sensation units.

The curves are not plotted above 4,000 cycles, since for frequencies above this value no consistent correlation exists between the results obtained from the two audiometers.

It seems to me that an interpretation different from that given by Dr. Bunch could very well be made from some of the charts given in his paper. For example, in his discussion of chart 4, the point is made that the dip is found at 4,700 cycles by one audiometer and not by the other. In this connection, I desire to point out that there are other differences just as large between the two curves in this high frequency region, which can be explained only by variations in the apparatus, in the testing technic or in the patients' judgments. For example, at a frequency of 8,192, the "A" audiometer gave results indicating that the left ear was 35 SU better than the right ear, which means that it would require 3,000 times more sound power to sense the tone in the right ear than in the left ear. In spite of this large difference shown by the "A" audiometer, the "B" audiometer indicated that the patient heard much better with the right ear.

The following simple experiment is suggested as a means of determining which of these two curves is correct.

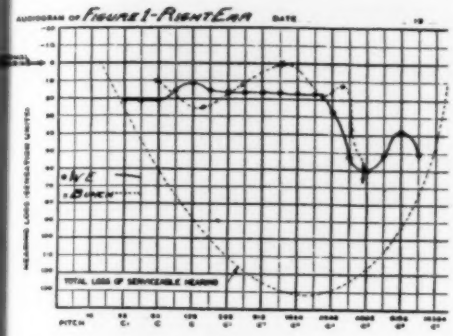
Using either audiometer, adjust the tone until the receiver is giving a pitch of 8,192 and an intensity near the threshold for one ear. Then suddenly transfer the receiver from one ear to the other. Differences as large as those shown on the chart will sound like large differences under such circumstances.

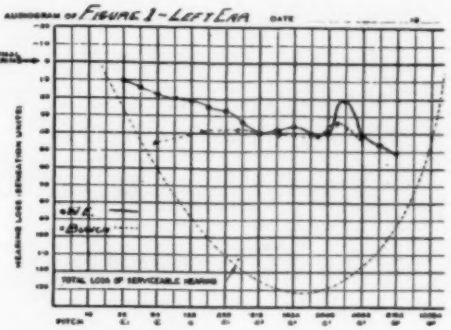
It is my understanding that the telephone receiver used with the "B" audiometer was undamped. Our experience in Bell Telephone Laboratories has indicated that any undamped receiver is considerably variable at these high frequencies. The special damped receivers for the "A" audiometer were designed by engineers having many years of experience in this field. Even these receivers are slightly variable for the high frequencies. Because of the variability of receivers at high frequencies, I would attach little significance to discrepancies in the two sets of curves for frequencies above 4,000 cycles. It was for this reason that no attempt was made to find calibration curves above this frequency.

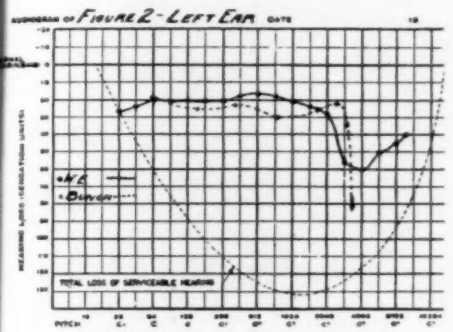
In discussing chart 5, Dr. Bunch concluded that the "A" curve was in error because it failed to agree with some voice tests which were made. From the data given, I would conclude just the opposite for the following reasons: Since most of the loudness in a whispered voice is carried by frequencies above 1,000 cycles, the "A" chart would indicate that for the whispered voice, the left ear would be more acute, which is in agreement with the whispered tests recorded. The loudness of the spoken voice is carried largely by frequencies between 500 and 2,000 cycles. Over this range, both charts would indicate about equal acuity for voice, which was found to be the case within the observational error.

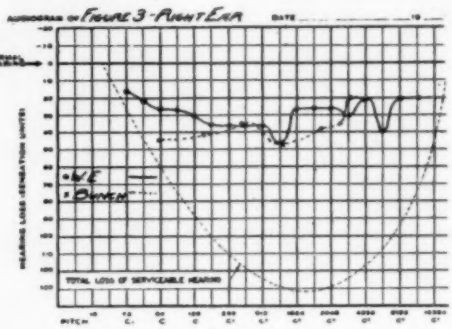
The differences shown in the other charts are probably insignificant, except as pointed out by the author.

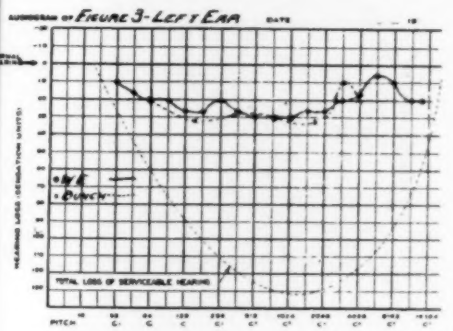
REDRAWINGS OF AUDIOGRAMS TO SHOW CALIBRATION CURVES
BY NEW SCALE

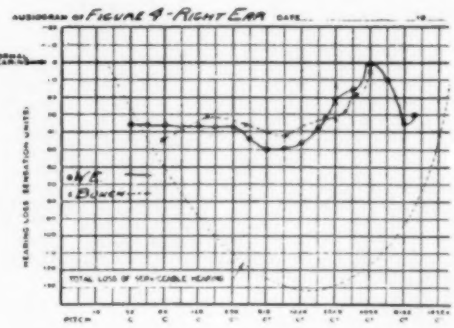


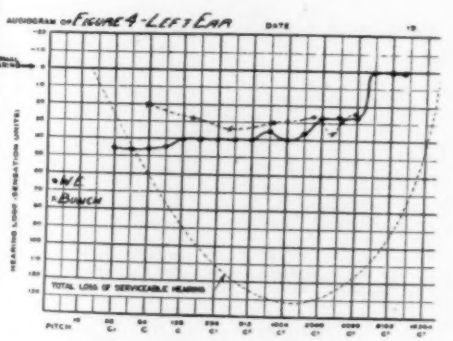


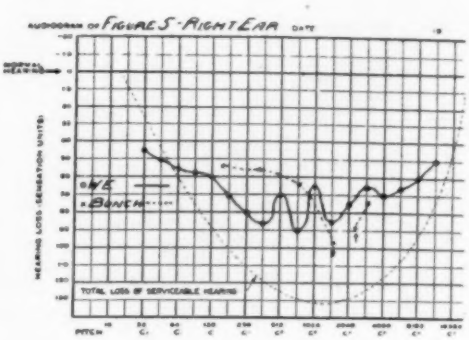




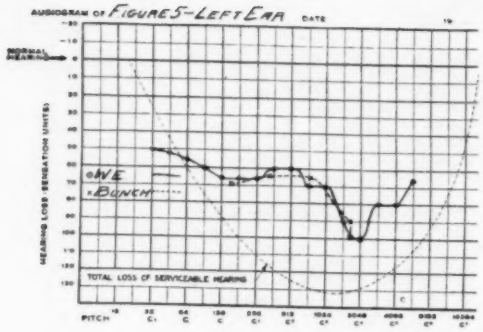




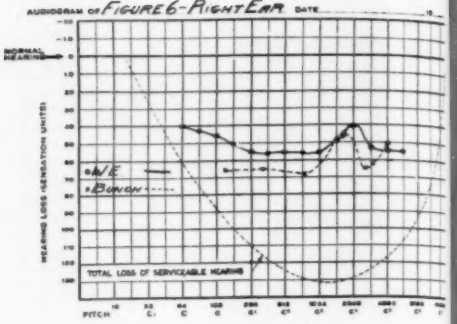




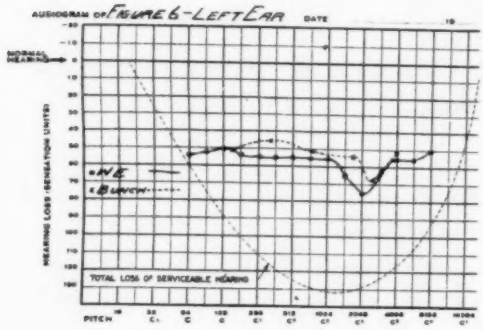
AUDIOGRAM OF FIGURE 5 - LEFT EAR DATE 19



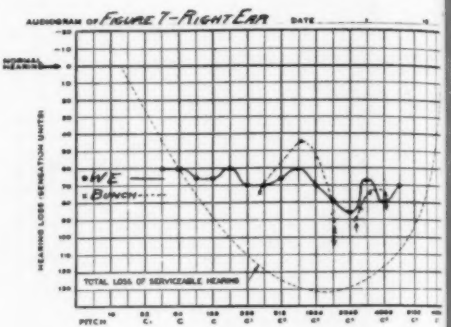
AUDIOGRAM OF FIGURE 6 - RIGHT EAR DATE 19



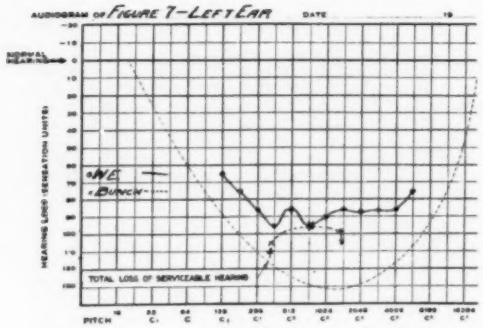
AUDIOGRAM OF FIGURE 6 - LEFT EAR DATE 19



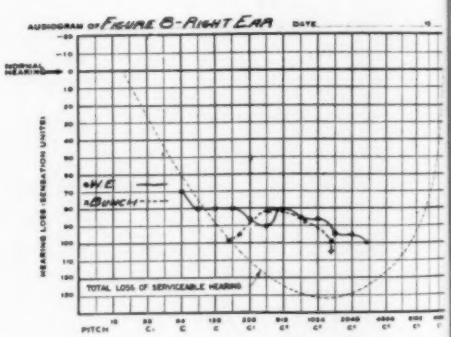
AUDIOGRAM OF FIGURE 7 - RIGHT EAR DATE 19



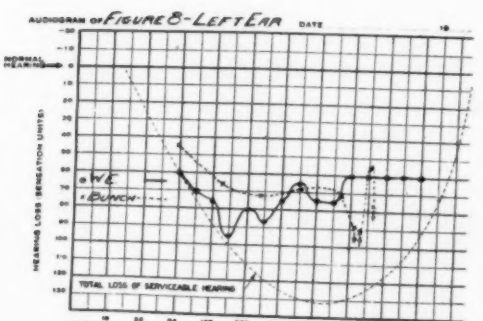
AUDIOGRAM OF FIGURE 7 - LEFT EAR DATE 19



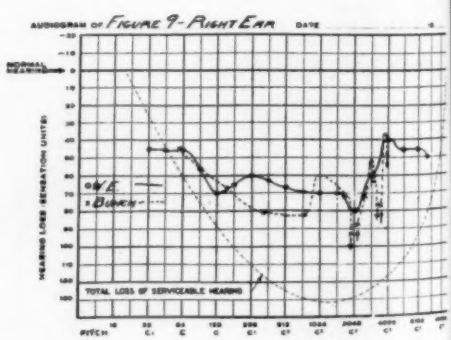
AUDIOGRAM OF FIGURE 8 - RIGHT EAR DATE 19

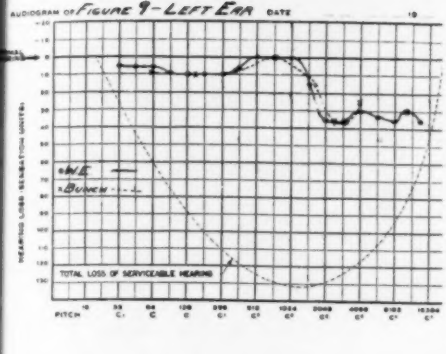


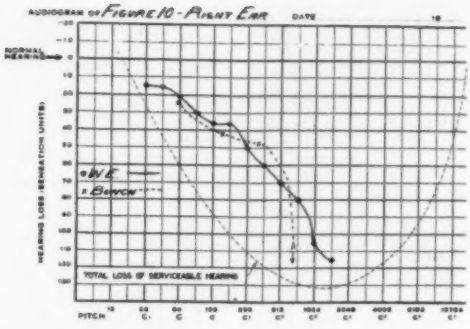
AUDIOGRAM OF FIGURE 8 - LEFT EAR DATE 19

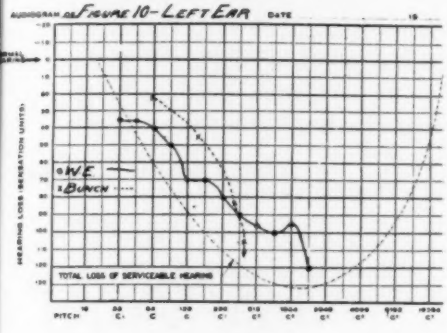


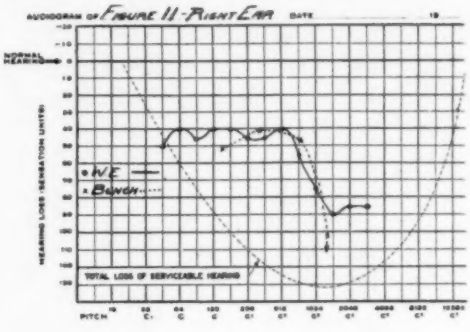
AUDIOGRAM OF FIGURE 9 - RIGHT EAR DATE 19

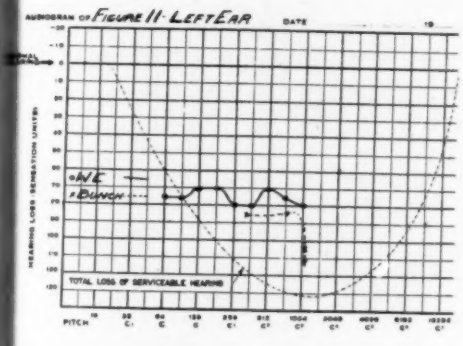


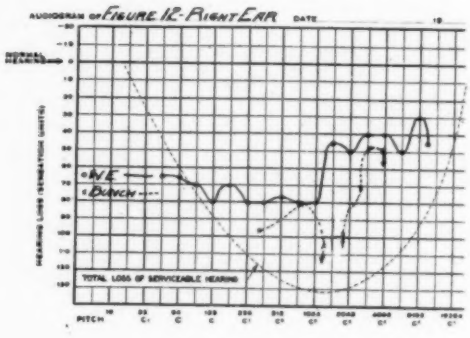

AUDIOGRAM OF FIGURE 9 - LEFT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

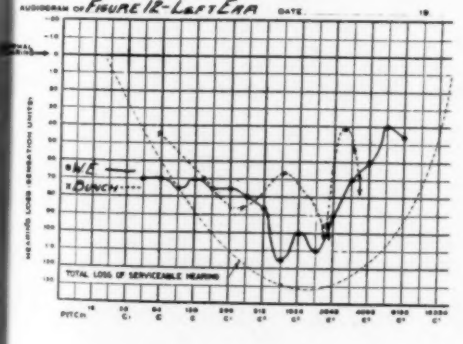

AUDIOGRAM OF FIGURE 10 - RIGHT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

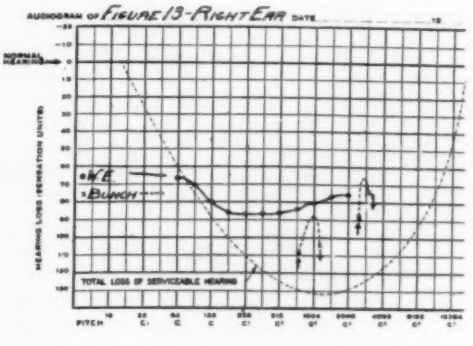

AUDIOGRAM OF FIGURE 10 - LEFT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH


AUDIOGRAM OF FIGURE 11 - RIGHT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH


AUDIOGRAM OF FIGURE 11 - LEFT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH


AUDIOGRAM OF FIGURE 12 - RIGHT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH


AUDIOGRAM OF FIGURE 12 - LEFT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH


AUDIOGRAM OF FIGURE 13 - RIGHT EAR DATE 19
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
•WE
×BUNCH
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

BILATERAL JUGULAR RESECTION FOR BILATERAL SIGMOID SINUS THROMBOSIS (OTITIC)

REPORT OF CASES *

HILL HASTINGS, M.D.
LOS ANGELES

In a search of the literature, I have been unable to find a report of a case in which recovery occurred after a bilateral sinus and jugular resection for septic thrombosis. The statement has been made by several authorities that a bilateral jugular resection would be fatal, due to the lack of sufficient return flow of blood from the brain. The fortunate result in my case indicates that such a procedure is not necessarily a fatal one.

REPORT OF CASE

W. A., a boy, aged 8 years, was seen on Jan. 2, 1925. Seven days prior to examination he developed a severe grip-like cold, with pain in the left ear. After three days, rupture occurred with drainage of thin, yellow pus. This boy had been operated on by me nine months previously for acute mastoiditis, with complete recovery and with normal tympanic membrane and normal hearing. The old scar became elevated, red and tender and the temperature rose to 102 F. Examination showed a considerable amount of pus in the left ear canal; the left mastoid was swollen and tender. The lungs and heart were normal. Urinalysis showed a slight trace of albumin, but no sugar or no casts. The right tympanic membrane was red and slightly bulging.

Operation was immediately performed on the left mastoid. The findings showed subperiosteal pus; the mastoid cavity (resulting from the previous operation) was in part filled in around the tip and above and behind the antrum. The remaining portion of the mastoid cavity was full of pus, and there was softened bone around the aditus. The lateral sinus was exposed at the knee, but was apparently not thrombosed. The dura above the antrum was exposed; its capillaries were slightly engorged. The pus in the mastoid cavity was decidedly thick and fibrinous, suggesting a pneumococcus infection. The right drum membrane was inflamed and bulging and was incised, with blood stained serous drainage. Cultures from the mastoid pus showed pneumococci.

The patient made a good recovery up to the seventh day; the temperature gradually dropped from 102 to a maximum of 100 F., rectal, on the fourth day; it was normal on the sixth day. In the afternoon of the seventh day, the patient had a chill, with rapid elevation of temperature to 104, with some drowsiness. Examination showed no apparent intracranial trouble and no labyrinthine signs or symptoms. Examination of the chest (by Drs. Dietrich and Berkley, pediatricians), however, showed a pneumonic process in the whole of the right lower lobe, with rough breath sounds over the entire right lung. The temperature went up to 105, rectal, thereafter gradually subsiding, reaching normal within six days. The patient was discharged in good condition thirteen days after the

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* Read before the Section on Laryngology, Otology and Rhinology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

* Osler, W.: Practice of Medicine, New York, D. Appleton & Co., 1905, p. 83.

operation. The left mastoid was granulating well, and the discharge had ceased. The right ear discharge was persisting; the right mastoid was not tender. The lungs were normal.

The patient was returned to the hospital ten days later, January 25, because of increasing tenderness and slight edema over the right mastoid and with an elevation of temperature to 101 F. in the afternoon. There were no other signs or symptoms except headache. The boy's general condition was excellent; there were no chills or sweating. The patient was operated on with the feeling that there was nothing indicated but a simple mastoid operation on the right side. The left mastoid wound was practically healed, with normal hearing in that ear.

During the operation on the right mastoid, to our great surprise we found destruction within the mastoid out of proportion to the signs and symptoms. There was some subperiosteal pus. The mastoid cortex showed cortical perforation with a large amount of pus escaping, apparently under pressure, pulsating. The mastoid structure had been entirely destroyed; the inner table over the lateral sinus above and below the transverse portion and in front of the vertical portion of the sinus was destroyed. The dura was covered with a fibrinopurulent exudate that did not wipe off completely, suggesting the same pneumococcus infection that was found on the left side. (Culture showed pneumococci.)

In the absence of any diagnostic symptoms, the lateral sinus was not opened.

The specimen of urine, taken before operation, showed about 5 per cent of sugar. The presence of this sugar (which had not previously been found) was explained by a consultant who was doing diabetic work, as an acute toxic glycosuria from the pus infection. The following day, examination by a neurologist (Dr. Carl Rand) showed that the reflexes were normal; there were no signs of intracranial trouble except slight haziness of both optic disks with rather full veins and vessels indipping in the optic cup. Lamina cribrosa not well seen. Ophthalmologic examination (Dr. Maghy), January 28, showed vision in the right eye was 20/20. The externals were negative, except that the pupil was widely dilated. The reaction to light and in accommodation and the consensual reactions appeared normal. The nerve head as a whole appeared larger than normal, especially in the horizontal diameter. The margins were slightly blurred, but there was scarcely any elevation. The veins were very tortuous and distended, while the arteries appeared about normal in size. There were no retinal hemorrhages in any part of the fundus. The lamina cribrosa was clearly defined. Vision in the left eye was 20/25. The externals were negative, except that the pupil was widely dilated. The reaction to light and in accommodation and the consensual reactions were quite normal. The nerve head was decidedly more blurred than in the right eye. The veins were very tortuous and distended. The arteries appeared normal. There was some edema extending into the retina which surrounded the optic disk, and at least 2 to 3 diopters of elevation. Adjoining the nerve head, one small hemorrhage was present, less than a disk diameter in size, situated below and to the temporal side. The lamina cribrosa was well defined.

The temperature the day following operation was 102 F. maximum, and for nine days thereafter did not exceed 102 except once, on the ninth day, it was 102.6. The minimum temperature was not under 100, except once on the second day. There were no chills or sweats. The sugar in the urine remained about 5 per cent in spite of careful feeding. The edema of the optic disks gradually increased, worse on the left side, and after three days small hemorrhages were noted in the right disk. The elevation of each disk was 1½ diopters. The headache persisted over the left frontal region. There was no vomiting or dizziness; no dulness or stupor, no chills, no intracranial symptoms to corroborate the eye findings and the sugar in the urine. The right mastoid cavity granulated slowly, the yellow fibrinopurulent exudate slowly being replaced by good granulations. Blood count January 27 (two days after operation) showed: leukocytes, 14,500; polymorphonuclears, 74.5 per cent; large and small lymphocytes, 21.5 per cent; transitionals, 3 per cent; eosinophils, 5 per cent; basophils, 0.5 per cent. Blood counts during the following few days were about the same. Blood culture, February 3, was sterile.

February 4, ten days after the right mastoid operation was performed, the temperature went up to 104 F. rectal. Definite swelling and tenderness over the upper part of the right side of the neck appeared along the course of the jugular. The appearance of this sign for the first time made us suspect a sinus and jugular thrombosis. So far, we had been more on the lookout for brain abscess because of the presence of sugar in the urine and the choked disks.

At the third operation, February 5, sinus and jugular explorations were done on the right side, the eleventh day after the primary right mastoid operation. (At this time the left mastoid was firmly scarred; the hearing was normal, and there was no reason to suspect any trouble on the left side.) On reopening the right mastoid, the dura around and over the sinus was so thick it was with difficulty that the sinus wall was outlined. The wall was very thick; the lumen apparently closed. The sinus wall was incised and a groove director passed backward, as well as below, toward the tip failed to develop bleeding. The neck was immediately opened at my request by a general surgeon (Dr. Sidney Burnap) and the vein was found thick, firm and thrombosed down to the level of the facial; below that it was normal. The vein was ligated below the facial and was resected to the extent of 1 inch, and the upper cut end of the jugular was brought to the surface and sewed to the cut edge of the wound. The thrombus within the vein was firm and hard and was not broken down into free pus. Cultures of the jugular thrombus showed staphylococci.

At the end of the operation on the sinus and jugular, an intravenous injection of insulin was given. The patient left the operating table in fair condition except the pulse rate was high, 150.

February 6, the day after operation, the temperature was 99.8 F., rectal; the pulse was 130; there were no meningeal signs, and the fundi showed a haziness in both disks; full veins; no new hemorrhages in nerve field; 1 diopter elevation (Dr. Maghy). We felt that the findings of the thrombus in the sinus and jugular were sufficient to account for the patient's signs and symptoms and that recovery could be expected. The presence of headache on the opposite side (left frontal) and changes in the fundi were disturbing symptoms and signs.

February 21, sixteen days after ligation of the right jugular, the patient had not done well. The left frontal headache persisted, which was very severe at times; drowsiness gradually increased; the patient slept practically all the time but when aroused was conscious. Five days before, two small fresh hemorrhages appeared in the fundus of the left eye. The elevation increased to 2 diopters. Neurologic examination by Dr. Rand was as follows: He was unable to elicit any deep reflexes; grip was about equal; the Kernig sign was suggestive but was not positive; there was no ankle clonus, and Babinski's sign was not present; lumbar puncture showed markedly increased spinal fluid, not turbid, clear, without increase in the cellular contents.

Sugar in the urine persisted in spite of injections of insulin and careful feeding. The temperature for sixteen days following the right sinus and jugular operation gradually decreased, and did not exceed 102 F., rectal, but once reaching 102.8 F., rectal; for three days it did not exceed 99.8, rectal, the lowest being 98. The pulse rate gradually decreased, keeping between 90 and 110. The chest and abdomen were normal. Frequent examinations for brain abscess were negative. There was slight puffiness of both eyelids.

In spite of the encouraging temperature chart, the patient was rapidly growing worse, with marked drowsiness and severe headache on the opposite side (left), and with increasing optic neuritis. On February 21, tenderness and slight thickening appeared along the upper part of the left jugular. (The left mastoid had been healed for several weeks, with normal hearing.) The parents were persuaded to permit me to open the left side, with the hope that we might find a sinus and jugular thrombosis on the left side similar to that which was found on the right, or that we might find an abscess on or within the brain on the left side.

February 22, an exploratory operation on the left side was performed. The findings were as follows: The left mastoid scar had healed; the mastoid cavity was filled in with scar tissue; the sinus bony groove was intact; the tegmen antri was not necrosed. The lateral sinus was uncovered, the wall was found

thick, the sinus did not appear definitely thrombosed, but had a boggy feel. The dura over the cerebrum and cerebellum was uncovered looking for an epidural abscess, but nothing was found. The lateral sinus was then explored and found thrombosed. The thrombus had partially broken down near the knee, and a small amount of yellow pus was wiped out. On removal of the thrombus free bleeding occurred from the upper end but none from below. The bleeding from above was not as free as one wished, but the wall was thickened and the vein partially collapsed. The left internal jugular was at once exposed and found thrombosed down to the facial vein and thickened slightly for one-third of an inch below the facial. The jugular was tied 2 inches below the entrance of the facial and resected; the upper end was brought out to the skin and sutured. A glucose injection was given while the patient was on the operating table. (Rapidity of the neck surgery, done at my request by Dr. Sidney Burnap, contributed considerably to the recovery.)

February 23, one day after operation of the sinus and jugular, the patient was in good condition, and clear mentally; the headache disappeared; there was no vomiting; edema of the eyelids and face was more marked. Eye examination by Dr. Maghy showed that the right eye had more edema and elevation than on any previous examination. Nerve head margins everywhere were indistinct with one quite large hemorrhage adjoining the disk at the temporal margin. The veins and arteries were very tortuous; there was elevation of 3 diopters; edema was still present at the temporal margin of the disk but no hemorrhage; vision was 20/200. The left eye showed less edema, and no fresh hemorrhages; the margin was still quite indistinct; there was less elevation; the vision was 20/200.

March 1, the right eye vision was 20/200, with an elevation of 3 diopters; there were no fresh hemorrhages. The vision in the left eye was 20/200; there was less edema.

March 10, marked improvement was seen in the optic disks; the vision in the right eye was 20/70, with an elevation of 1 diopter. In the left eye, the vision was 20/50, with no edema.

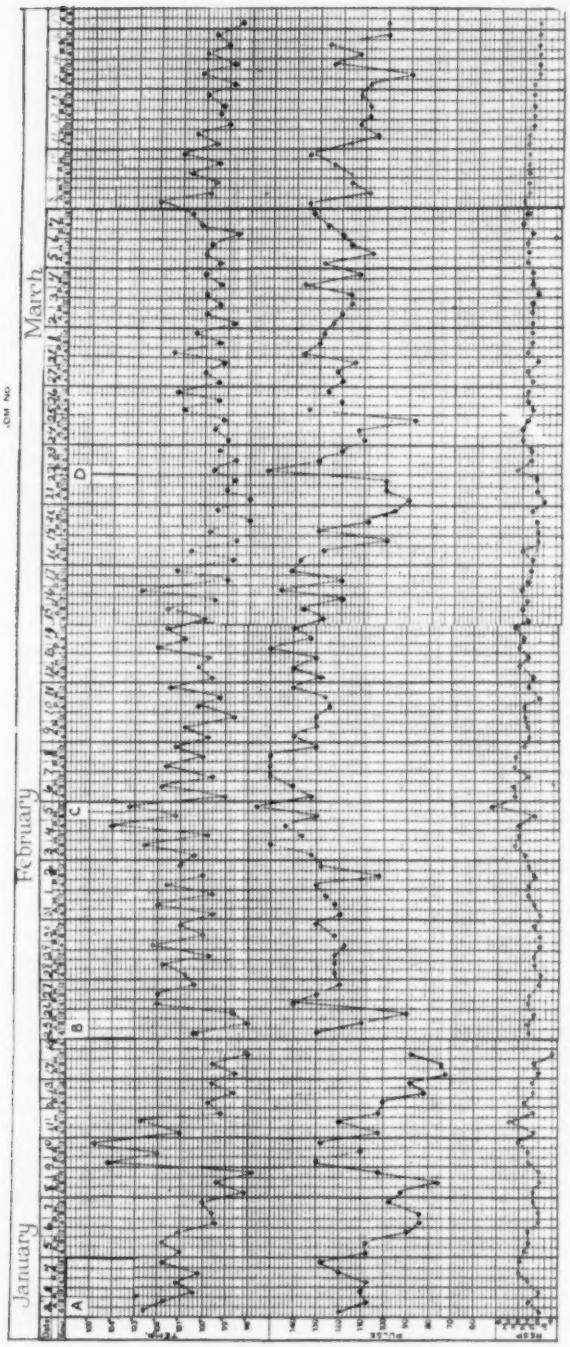
On March 15, three weeks since the left sinus and jugular operation, the patient had been improving slowly but steadily. The temperature after operation was 101 F., rectal, on three occasions; there were no chills and no sweats. For the past week the highest temperature was 101, rectal; the lowest 99, rectal. The pulse rate was from 110 to 140. The eyelids were still puffy; the mental condition was excellent. Drowsiness had entirely gone, and the patient slept and ate well, making a good recovery. He complained of a tender spot on the top of the head. Examination showed a swelling in the midline at the junction of the occipital and parietal sutures; thick, firm, apparently subperiosteal, about 3 cm. in diameter.

March 17, the swelling at the top of the head had slightly increased without much tenderness. The temperature for two days was not over 99.8, rectal; the condition of the patient was excellent. At operation, performed by Dr. Carl Rand, the subperiosteal abscess at the occiput was opened under gas-ether anesthesia. The findings were, enormous distention of the veins of the scalp (evidently collateral circulation changes, the result of double sinus and jugular thrombosis). The subperiosteal abscess was opened and a teaspoonful of pus was evacuated. The cranial plate was firm, not carious so far as could be determined; the bone was not removed. Culture of the pus on blood agar plate showed pneumococci.

March 20, the patient was much better. Ophthalmoscopic examination by Dr. Maghy showed vision in the right eye, 20/40; the margins were indistinct. The color of the nerve head was grayish red; the vessels were small. In the left eye, vision was 20/40, the margins still were not clearly defined; the color appeared normal.

The patient made an excellent recovery from the last operation. Drains were removed at the end of ten days and the wound healed promptly. Both mastoids healed with normal drum membranes; the hearing was normal.

The cause of the subperiosteal abscess noted in the foregoing was not determined. It is believed that it was due to an embolus or else resulted from a thrombus at the occipital end of the sigmoid sinus of one side. (It should be



Temperature Chart.—*A*, left mastoid operation; *B*, right mastoid operation; *C*, right sinus jugular operation; *D*, left sinus jugular operation.

remembered that there was not good bleeding from behind on opening the right lateral sinus; and on removal of the thrombosis in the left lateral sinus, only fair bleeding from behind.)

May 1, six weeks after the last operation, the temperature had remained normal; there were no headaches, and edema of the eyelids had entirely disappeared. The patient gained in weight, and felt well in every way. Examination of the eyes by Dr. Maghy showed that the fundi were almost normal; there were no edema and no hemorrhages. Vision in the right eye was 20/25; in the left, 20/30. Both ears were normal.

November 24 (seven months later) the patient has been under constant observation. He has been entirely well in every way. The urine remained sugar free. Ophthalmoscopic examination by Dr. Maghy showed that vision in the right eye was 20/15; the margins were quite distinct, except for a small part of nasal border; the color was normal; the markings of lamina were entirely obscured. Vision in the left eye was 20/20 plus 4; the margins were fairly well defined, except for a small area on the nasal side. Lamina cribrosa was entirely obliterated. There were no color scotomas.

April 4, 1926, a year since recovery, the patient reported for observation. He has remained in perfect health.

COMMENT

In the history of my case, certain points are worth emphasizing:

1. The absence of chills and absence of markedly septic temperature. The second sinus and jugular were explored and found thrombosed (seventeen days after the sinus and jugular resection of the other side) in spite of the fact that the temperature had been approaching normal for several days, without any chills or marked remissions of temperature. It is evident that simultaneous jugular occlusion by thrombosis had existed for some time prior to the resection of the second internal jugular.

2. The severe headaches and drowsiness, the edema and hemorrhages in both optic disks (which appeared early), and the glycosuria had led us to suspect a brain abscess rather than a sinus thrombosis. In reoperating on the opposite side, the sinus thrombosis was discovered by an exploratory operation on a mastoid that had been healed for three weeks with normal drum membrane and normal hearing. The brain was, therefore, not opened.

The occurrence of a subperiosteal abscess of the occiput should be emphasized. We believed that this was due to infection from within the cranium through a thrombus at the torcular. At the time of opening the right sigmoid sinus it was impossible to secure free bleeding from the right transverse sinus although resected to a considerable extent toward the torcular. Although the roentgen-ray picture showed a dark area 2.5 cm. in diameter, the bone underlying the subperiosteal abscess was not necrotic and was not removed. Healing occurred promptly.

REVIEW OF THE LITERATURE

In the search of the literature we found one case in which a torcular subperiosteal abscess occurred, and to such an extent that is worth quoting.

A. L. Whitehead [1] reported a case of thrombosis of the right lateral sinus, in which the clot extended beyond the torcular into the lateral sinus of the opposite side. An operation was performed, with recovery. The patient had had right otorrhea for five years, and acute illness, seven days. The optic disks were congested; there was blurring of the edges. A right radical mastoid operation disclosed pus and caries, and an epidural abscess. Two days later the sinus was exposed because of persistent elevation of temperature. It was thrombosed to and over the torcular; the openings of the longitudinal sinus and the sinus of the other side were blocked with firm clot; a small curet, passed into the opposite sinus, failed to set up bleeding. The internal jugular on the right, which was collapsed, was tied. During the next three days there was progressive edema of the opposite side of the face; the superficial veins were greatly dilated; the temporal, facial and external jugular appeared about the size of a healthy internal jugular. There was intense headache but no definite optic neuritis. Five months after the first operation, fluctuation developed under the scalp and a quantity of pus was evacuated; between the scalp and the bone a mass of breaking-down granulation tissue was found, which was thoroughly scraped away. The abscess was under the periosteum; the surface of the bone was rough, pitted and exposed from the orbits to the occiput, and laterally to the upper border of the mastoid process. For many weeks healing took place without any necrosis of the skull. Slow, uneventful recovery followed.

In a discussion of the case, C. Ernest West stated that he had seen a boy in whom both jugular veins had been tied without edema resulting until late in the course of the case, and that as the cavernous sinus had then become blocked it must have been the obstruction to the orbital and facial veins which caused the facial edema.

In our search of the literature we were able to find five cases reported by general surgeons in which both internal jugulars required ligation during operations for cancerous or tuberculous conditions. These cases have been reported by Baldwin,[2] Ewald,[3] Dangel,[4] Duval [5] and LeGrand.[6] None of these, however, were of aural disease.

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1. Whitehead, A. L.: Thrombosis of Lateral Sinus Extending Beyond Torcular; Occipital Subperiosteal, Brit. Roy. Soc., Otological Section, December, 1908.

2. Baldwin, J. F.: Synchronous Ligation of both Internal Jugulars, Ann. Surg. **36**:394, 1902.

3. Ewald, C.: Tod unter vasomotorische Storungen nach Resektion bieder Venae jugulares internae, Wien klin. Rundschau. **14**:673, 1900.

4. Dangel, Max: Ueber die Unterbindung den Vena jugularis interna. Ein Fall von doppelseitiger Unterbindung, Beitr. z. klin. Chir. **16**:495, 1905.

5. Duval: Ligature des deux jugulaires internes, Bull. et mém. Soc. de chir. de Paris **35**:224, 1909.

6. LeGrand: Un case de ligature bilateral des jugulaires internes, Normandie méd. **33**:58, 1922.

Baldwin's case was accidental, seen in connection with removal of cervical tuberculous glands. He stated that in the literature at his disposal, he found no case of bilateral jugular resection with recovery. He did not observe any profound cerebral disturbance following the operation. One ligation followed immediately the ligation of the other internal jugular. Recovery was uneventful except on the third day; on dressing the wounds there were convulsive seizures lasting about twenty seconds.

In Ewald's case, the bilateral jugular resection was performed in connection with an extensive neck operation for cancer of the pharynx, tongue and cervical glands. The patient died before regaining consciousness.

In Dangel's case, both internal jugulars were resected during extirpation of carcinomatous lymphatic glands. No brain disturbance and no eye symptoms were observed to follow the simultaneous bilateral resection. There was, however, marked cyanosis for about ten seconds after tying the second jugular. The patient recovered from the operation but died four weeks later from erysipelas.

In Duval's case, operation was done for extirpation of cancerous cervical glands. Both vessels were ligated at the same operation. No brain disturbance and no eye symptoms or signs resulted, although there was cyanosis of the face noticed for a few hours. The patient recovered.

In LeGrand's case, the original report is not available. There is no reason to believe that it was of aural origin.

It is evident from these reports of general surgery that bilateral jugular resection is not necessarily a fatal procedure, contrary to the impression of many otologic authorities. When bilateral sinus thrombosis is found, with every reason to believe that there is a septic thrombus in each of the internal jugulars, the brain has already been subjected to the limited return through collateral venous channels. To ligate the jugulars should not be an added risk; on the contrary, a life may be lost by failing to do this.

In a recent letter, one of my confrères states that in a case of his own in which bilateral jugular thrombosis was suspected, he discussed the matter with the professor of anatomy at his university, who was firm in his belief that both internal jugulars should not be ligated at the same time. His patient died without bilateral resection. No necropsy was obtained.

In the search of the literature, we were able to find only six cases in which recovery took place after resection of one jugular in connection

with bilateral sinus resection of otitic origin. These cases are Gruening's,[7] C. H. Smith,[8] O. D. Stickney's,[9] H. G. Tobey's,[10] Dausend's,[11] Schlegel's.[12] In only two of these cases were choked disks noted.

As to the collateral circulation after bilateral jugular resection, Tobey [10] quotes Ballance, who in turn quotes from Charpy:

> Under normal conditions, almost the whole of the blood from within the cranium is returned by the two internal jugular veins; there are, however, numerous anastomoses between the intracranial and the extracranial venous circulation, which allow of alternative routes when one or the other of the jugular veins is not available.
>
> There are three principal and many accessory communications. The three principal are: 1. The plexus of the foramen magnum, the uppermost of the spinal plexuses, anastomoses with the transverse occipital sinus in front and with the posterior occipital sinuses behind. Further, these plexuses, or their collecting trunks, the vertebral and posterior jugular veins, anastomose with the posterior condyloid emissary vein and with the mastoid emissary vein. The communications here are so free that the whole of the blood from within the skull can emerge by this route after ligature of both jugulars. In the fetus and even in the new-born, the cranial surface of the lower half of the occipital bone is covered with a close network of large veins, which take up the blood from the torcular and its vicinity and discharge it into the origin of the jugular. As development proceeds, this network becomes gradually changed into the occipital sinuses; hence, the great variety in the size and arrangements of these vessels. 2. The ophthalmic vein and its affluent, through which a considerable volume of blood may enter the facial and temporal veins. 3. The middle meningeal veins, through which there is communication between the superior longitudinal sinus and the pterygoid plexus.
>
> The accessory communications are innumerable, for the origins of the external jugular and of the facial portion of the internal jugular communicate with those of the intracranial veins practically everywhere over the vault and over the base.

This was well seen in our case, due to a complete shaving of the head on reoperating for the subperiosteal abscess at the occiput. The veins over the cranium and front of chest were enormously enlarged.

Kloestadt [13] says:

> Blocking of one lateral sinus is not likely to cause any intracranial circulatory symptoms. There are cases, however, where one transverse sinus is very small —usually the left; or the superior longitudinal sinus passes entirely to the right side without any anastomoses at the torcular. There is a correspondingly large right and a very small left internal jugular vein. Patients with this anatomical pecularity are likely to develop intracranial symptoms and may die if the larger venous channel is occluded. This applies with still greater force to those rare cases where both internal jugulars have to be tied.

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7. Gruening, E.: Case of Bilateral Thrombosis of Sigmoid Sinus. Recovery. Tr. Am. Otol. Soc. **9** and **10**:518-520, 1905-1907.

8. Smith, C. H.: Double Mastoid with Septic Sinus Thrombosis, New York M. J. **101**:407, 1915.

9. Stickney, O. D.: Bilateral Acute Suppurative Otitis Media with Symptoms of Sinus Thrombosis, Laryngoscope **29**:90, 1919.

10. Tobey, H. G.: Double Acute Suppurative Mastoiditis with Symptoms of Sinus Phlebitis or Thrombosis, Tr. Am. Laryng., Rhinol. & Otol. Soc. **29**:6, 1923.

11. Dausend, J. A.: Ein Fall doppelseitiger Sinus thrombose, Beitr. z. Anat. Physiol., Path. u. Therap. d. Ohres. **4**:394, 1910-1911.

12. Schlegel, G.: Ein Fall von doppelseitiger Sinus thrombose, mit ein sitige jugularis Unterbindung, Arch. f. Ohrenh. **69**:176, 1906.

13. Kloestadt: Ztschr. f. Laryngol. November, 1924.

In the light of the case I have reported, and from a study of the literature, it would seem that much of the fear of bilateral ligation is unwarranted.

ABSTRACT OF DISCUSSION

Dr. George M. Coates, Philadelphia: Doubtless the first time this procedure was carried out, it was done with a great deal of trepidation; since then it has become quite common. For a long time discussion was rife as to what would happen if two sinuses were infected at the same time; how we could make the diagnosis; what would happen if we made a wrong diagnosis, operated on the sinus that was not infected, and had to operate on the infected sinus. We know that both sinuses can be obliterated by a thrombus, and further obliterated by operation, and there will be no serious after-results. Dr. Hasting's very interesting case shows that we can do even more than this, and ligate farther down the neck, obliterate the internal jugulars, and thereby cut off the greater portion of the return circulation to the brain. It seems to me that the salient point in this case is the fact that, first, he has ligated these veins; second, that before ligation of the veins the circulation was cut off so that the ligation did not make any great difference; and third, that the collateral circulation had probably ample chance to become established through the slow development of the thrombi. If both jugulars had been tied off at once it seems to me the outcome might have been different; but with the slow development of the thrombus, one side being shut off first and later the other one, the collateral circulation had ample chance to develop.

Dr. O. Jason Dixon, Kansas City, Mo.: I am opposed to the ligation of the internal jugular for sinus thrombosis. In Dr. Hasting's case, in my opinion, it was not proved that the thrombus on the left side would have caused any trouble if it had been left alone. The patient might have had septicemia, and that is all there is to it. I grant a complication in the brain, say an abscess, might have resulted and that if a thrombus breaks down it needs drainage, but tying off the internal jugular does not stop the septicemia. It simply adds another hazard for a very sick patient. The leukocyte count is, in my opinion, of very much value in the diagnosis of sinus thrombosis —not so much whether it is high or low, but making the count at the onset of the illness and later noting its up or down curve. Also, I am very reluctant to diagnose sigmoid sinus thrombosis without a clinical picture of chills, a septic temperature, a history of ear trouble, and a leukocytosis, the patient presenting a clinical picture of well-being, yet being very sick with a positive blood culture. This picture may result from something else besides sinus thrombosis. I have seen postoperative mastoid cases develop the clinical picture of sigmoid sinus thrombosis, but by waiting, the cause was malaria in one case, pneumonia in two cases, erysipelas in one case and endocarditis in one case. We must take these things into consideration. Radical surgery would be doing the patient a rank injustice. I have cases presenting the clinical picture of sinus thrombosis. I did not go into the sinuses. I simply assisted Nature to complete her task. I revived the overlying infected bone from the sigmoid and instituted drainage. All of these patients, except one responded promptly to this treatment. We know from our findings at operation that in nearly every case of streptococcic mastoiditis there is a sinus phlebitis. Rarely do these cases go on to thrombosis.

I believe that too frequently patients are operated on too soon after the onset of the mastoiditis. We must wait; stand ready to proceed with a radical operation, if necessary, but give the patient a chance to immunize himself. I have seen just as many patients continue their septicemia and wear out their infection with ligation of the internal jugular as without it.

In regard to intravenous therapy, I do not use it. These toxic patients must be kept saturated with fluids. If the patient continues to have chills, all you have to do is to slit the sigmoid that is already uncovered and remove

the infected clot. I believe that all of us will live to see the time when not even a general surgeon will tie off an internal jugular vein.

Dr. R. H. T. Mann, Texarkana, Ark.: I performed a mastoid operation on a child who developed a lateral sinus thrombosis. Ligation of the internal jugular vein was refused. Blood culture showed the infecting organism to be *Streptococcus hemolyticus*. Two intravenous injections of mercurochrome-220 soluble cured this child. I do not recommend the promiscuous use of this drug, but when all else has failed, its trial might not be out of place in well selected cases.

Dr. S. E. Roberts, Kansas City, Mo.: I had a case six years ago, in which both lateral sinuses were opened at the same time. My patient had a double mastoiditis, the right ear appearing to be the worse of the two ears. Both sinuses were thoroughly exposed at the first operation; both were covered with granulations. I did not open either sinus. I agree with Dr. Dixon it is better to await developments. The patient was better for three days, but on the fourth or fifth day she had a chill, followed by a high temperature. I did not know on which side the thrombus existed. The neurologist advised opening the right lateral sinus, but nothing was found. The sinus was packed firmly to control bleeding. I then incised the left sinus and found it completely thrombosed. I was unable to get bleeding from either end. No ligation was done. The child continued to have a mild septic temperature for a week, without chills. She made a complete recovery.

Dr. Hill Hastings, Los Angeles: Dr. Coates mentioned a very important point, that in the development of the thrombi an interval of time must have elapsed, probably a week or ten days, which gave the collateral circulation a chance to be established. When it comes to leaving thrombi in the veins, I have seen too many fatalities from failure to ligate below an infected thrombus. Furthermore, when it comes to waiting for the diagnosis of sinus thrombosis, until chills and sweats appear, you are liable to lose a patient. The old signs of sinus thrombosis were the signs of septic disintegration of the thrombi. If you wait for that to occur, three times out of four it will be too late. Most of our recoveries after all, are in cases in which the diagnosis of sinus thrombosis is made at the time of operation.

PROGRESS IN OTOLARYNGOLOGY

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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INTRADURAL COMPLICATIONS OF AURAL AND NASAL ORIGIN

A SURVEY OF RECENT LITERATURE *

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

THE RÔLE OF EAR AND NOSE IN INTRACRANIAL LESIONS IN GENERAL

Trauma.—A suppurating ear following a fracture through the petrous pyramid is the cause of death in about 8 per cent of cases of intracranial lesions, while a depressed fracture through one or other of the accessory sinuses gives rise to septic meningitis much more frequently—possibly months later—although a linear fracture through the anterior fossa of the base is usually exempt from meningitis.

Surgical treatment of meningitis from a complicating suppurative otitis or sinusitis gives a better prognosis in fracture cases than in primary otitis or sinusitis, because a protective zone of adhesions is built up in the pia-arachnoid prior to the advent of the suppurative process. Consequently, drainage of the infected area (if performed early) results in a cure in a considerable proportion of cases.

Rand and Nielsen [1] report seven cases of meningitis in 171 skull fractures. The portal of entry was through the ear in four instances, and through the upper nasal passages by way of the cribriform plate or ethmoid cells in two others.

One patient (mucosus capsulatus of Friedländer) recovered after intravenous injections of mercurochrome-220 soluble and repeated lumbar punctures.

These authors emphasize that a bleeding ear must never be irrigated, but the canal plugged with sterile cotton.

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* Reviewer's Note: In this survey I have attempted to apply the philosophy of intradural suppuration to the unusual and unexplained cases of recent literature whenever it is applicable; leaving the real advances to stand for themselves because of my lack of personal verification or experience, and often adding, I regret, a too scanty word of praise.

1. Rand, Carl W., and Nielsen, Johannes M.: Fracture of the Skull: Analysis of One Hundred and Seventy-One Proved Cases; Diagnosis and Treatment of Associated Brain Injury, Arch. Surg. **11**:434-458 (Sept.) 1925.

The Nasal Cavity and Accessory Sinuses as the Route of Nonsuppurative Infections in the Brain.—Bronzini,[2] under the title "Vascular Relations Between Subarachnoid Spaces and the Healthy or Inflamed Nasal Mucosa," points out that inflammatory conditions of the nasal mucous membrane prevent the drainage of the cerebrospinal fluid through the lymphatics of the mucosa.

According to Yates and Barnes,[3] the infection in lethargic encephalitis is possibly a symbiotic process. At first the nasal mucous membrane is damaged by one of the catarrhal group of organisms; the subsequently inhaled virus of encephalitis passes through the damaged mucous membrane, and is absorbed into the brain by way of the perineural lymphatics.

Disease of the Nose and Ear in the Etiology of Intradural Suppuration.—(*a*) Congenital Defects: That *congenital defects* in the sphenoidal sinus may be the explanation of the sudden appearance of meningitis after a coryza without suppurative sinusitis, is anatomically established by the work of Gilse,[4] who also found two cases in the literature (Meyers[5]) in which a prolapse of the dura mater into the sphenoidal sinus is recorded. All these defects occur at one developmental site.

(*b*) Abscess of Hypophysis: Cases of acute suppurative hypophysitis complicating purulent sphenoidal sinusitis have been reported by Harvey, Meyer, and Boggs and Winternitz. Glinski, Smoler and Thom and Plaut have reported abscesses in the anterior lobe of the hypophysis as an accompaniment of purulent leptomeningitis. Simonds[6] reviews them and adds two of his own, both following otitis.

Case 1.—Bilateral suppurative otitis media and mastoiditis in a boy were complicated by purulent leptomeningitis. Postmortem examination revealed an abscess about one-fourth the size of the entire lobe, beneath the capsule on the front of one lateral half of the anterior lobe of the hypophysis.

Case 2.—Acute suppurative otitis media was complicated by purulent leptomeningitis. Necropsy showed that almost one entire lateral half of the anterior lobe of the hypophysis had been destroyed by an abscess.

Simonds says that hypophysial abscesses, complicating purulent leptomeningitis, are not infrequent, and are interesting because of the mechanism by which the infection reaches the gland. They are most commonly situated immediately beneath the capsule in the anterolateral

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2. Bronzini, A.: Arch. ital di otol. **36**:824, 1925.

3. Yates, A. L., and Barnes, S.: Nasal Sinuses as Route of Infection in Lethargic Encephalitis, Lancet **2**:130, 1925.

4. Gilse, P. H. G. van: Investigation on the Development of the Sphenoidal Sinus, J. Laryng. & Otol. **41**:137-144 (March) 1926.

5. Meyers, A. W.: Ann. Otol., Rhin. & Laryng. **24**:257, 1915.

6. Simonds, J. P.: Studies on the Pathology of the Hypophysis: V. Abscess of the Hypophysis, Endocrinology **9**:117-121 (March-April) 1925.

quadrants of the anterior lobe. Here two fairly large arteries enter at a little distance from the sagittal plane. These vessels penetrate the glandular portion and pass to the rich plexus of vessels in the core of connective tissue which is located at about the center. The infection appears to enter the hypophysis along one or the other of these blood vessels, perhaps by way of the perivascular lymphatics.

Fink [7] also reports an abscess of the hypophysis, the result of a thrombophlebitis of the cavernous sinus from suppurative ethmoiditis and sphenoiditis.

(*c*) Brain Abscess and Bronchiectasis: Adam [8] discusses "The Connection of Brain Abscess with Bronchiectasis," and, after calling attention to the fact that abscess of the brain is a known cause of death in bronchiectasis, says the association has been puzzling, but it seems probable that the abscess may result (like the bronchiectasis itself) from a nasal sinusitis rather than be a metastasis from the lung. Corroboration of this view is afforded by his two cases of bronchiectasis with brain abscess; in both there was nasal sinusitis.

(*d*) Intracranial Pneumatocele: All recorded cases of intracranial pneumatocele are reviewed by Dandy,[9] who says that while there are two sources of extracranial aerogenous tumors, (*a*) the mastoid cells and (*b*) the frontal sinus; intracranial pneumatoceles receive air (1) from a break in the wall of the skull, through which this air is forced, or (2) by the product of gas producing organisms after their entrance into the brain.

In one of his cases the air entered through a fracture of the frontal bone; in the second, through a fracture of the temporal bone, and in a third, it probably followed an operative defect of the mastoid cells.

A man entered the hospital in a state of semicoma, with a temperature of 107.2 F., and a pulse rate of 110. One week before admission, he complained of severe headache and pain in the left ear. Repeated chills and stiff neck followed. The left mastoid was tender. The leukocytes numbered 12,000; polymorphonuclears, 85 per cent. The spinal fluid was not under marked pressure (70 mm. of water), but was of ground glass appearance and contained 2,000 cells (96 per cent polymorphonuclears); there were no organisms. Pressure on the left jugular vein caused a rise of spinal fluid to 140 mm. and on the right to 160 mm.

The mastoid antrum contained a collection of pus of foul odor; the mastoid abscess was walled off but communicated with an extradural abscess. From the pus three organisms were grown: (1) *Bacillus coli*, (2) *Staphylococcus albus* and (3) the pneumococcus, type IV.

A second collection of pus of a foul fecal odor, with many bubbles of gas, lay immediately beneath the dura.

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7. Fink, E. B.: The Intracranial Complications of Posterior Sinus Infections: Report of an Unusual Case with Autopsy Findings, Surg. Gynec. & Obst. **41**:689, 1925.

8. Adam, J.: J. Laryng. & Otol. **41**:93, 1926.

9. Dandy, Walter E.: Pneumocephalus (Intracranial Pneumatocele or Aerocele), Arch. Surg. **12**:1-982 (May) 1926.

The patient died three days later.

Necropsy showed a large abscess of the left temporal lobe with a fistula in the descending horn of the left lateral ventricle; diffuse meningitis; thrombosis of the left lateral sinus (presumably a recent development, since Queckenstedt's test on admission had indicated the sinus to be patent).

Dandy feels that the air was forced through the mastoid defect into the intradural abscess cavity by swallowing, coughing or sneezing.

His conclusions are: 1. Pneumocephalus may follow in the wake of any artificial communication between the paranasal or mastoid sinuses and the cranial chamber. The opening may result from a fracture, from an operative defect, from the erosion of a chronic infection, or from destruction of the floor of the skull.

2. Pneumocephalus also results directly from infections of gas producing organisms—*Bacillus aerogenes-capsulatus* (of Welch) and possibly the colon bacillus.

3. The source of air may be the frontal, ethmoid, sphenoid and mastoid sinuses.

4. The symptoms are mainly those of increased intracranial pressure. A discharge of cerebrospinal fluid is usually present; its occurrence should always cause suspicion of pneumocephalus. Sneezing is a frequent symptom. Rhinorrhea after sneezing or after change of position is almost pathognomonic of this condition.

5. In fistulas through the frontal sinuses, the dural tear if located may be covered by a transplant of fascia lata.

Differential Diagnosis in the Presence of Suppurative Otitis or Sinusitis.—Hofer [10] reports a case of tuberculous meningitis in a child, aged 2, combined with otogenic thrombophlebitis.

On admission, the child presented the typical picture of otogenic thrombophlebitis, with intermittent temperature and bilateral middle ear suppuration. Left sided trephining of the mastoid showed suppuration under pressure (undoubtedly the cause of the intermittent fever). The temperature curve became continuous. There was no rigidity at the nape of the neck or turbidity of puncture fluid, but an increase of the cellular contents, at first slight, later considerable. Necropsy showed extensive tuberculous meningitis and diffuse miliary tubercles in all organs of the body.

Schlander [11] emphasizes a case involving the difficulty of a decision as to the meningitis being otogenic or due to other causes when a middle ear suppuration is present.

The patient's appearance suggested a nonotogenic cause of the meningitis, which was confirmed by well marked rigidity at the nape of the neck, not usually

10. Hofer, J.: Monatschr. f. Ohrenh. **60**:80, 1926.

11. Schlander, E.: Cerebrospinal Meningitis in Bilateral Chronic Middle Ear Suppuration, Monatschr. f. Ohrenh. **60**:70, 1926.

seen in otogenic meningitis, and the absence of all signs of an acute exacerbation of the ear suppuration. On the other hand, the adherent crusts in the aural attic, the sudden onset of deafness and the negative findings in the cerebrospinal puncture fluid were suggestive of an otogenic complication. A bilateral mastoid operation was performed with negative findings. Spinal puncture fluid showed meningococci.

Influence of Trauma in the Liberation of Septic—or Nonseptic—Emboli into the Brain.—That embolic septic—or nonseptic—particles must frequently be thrown into the circulation by the trauma of an operation is a mechanical necessity; the importance of this has only recently received the attention it should (especially in infections of the valveless facial veins—when squeezing or incisions are frequently followed by fatal retrograde thrombophlebitis).

Di Vestea's [12] contribution from the University of Pisa refers to the case of a woman (aged 37) on whom an operation for acute mastoiditis was followed by an apoplectic seizure (hemorrhage of the internal capsule) four days later.

Aids to Differential Intracranial Diagnosis.—(*a*) Cerebrospinal Fluid: Ohnacker,[13] under "Investigations on the Value of Sugar Determination in the Cerebrospinal Fluid in Otological Practice," says that the diagnostic value of the ratio of blood sugar to the sugar contents in the cerebrospinal fluid, for the differentiation of brain abscess, brain tumor, sinus thrombosis and meningitis, has not yet been investigated sufficiently. In hyperacute otogenic meningitis, the sugar content was diminished in 86 per cent of his cases, normal in 14 per cent, but never increased. In meningitis of a slower course, the corresponding proportions were 31, 33 and 6 per cent, respectively. A fairly constant diminution was noted only in very acute cases of meningitis.

Karbowski [14] studies the "Prognostic Value of Spinal Puncture in Intracranial Oto- and Rhinogenic Complications." He reports forty-five cases; of which thirteen were cases of cerebral abscess, three of intrameningeal abscess and seventeen of meningitis. Four patients with turbid fluid had meningo-encephalitic abscess, the suppuration having extended by continuity from the mastoid process to the brain. In 69 per cent of the brain abscess cases the fluid was clear; consequently, Karbowski thinks that a clear fluid is characteristic of brain abscesses. This, however, adds to the difficulty of the differential diagnosis between brain abscess, tuberculous meningitis and syphilis. Of the three cerebellar abscess cases, the fluid was turbid in two and clear in one. The same applies to intrameningeal abscess. Of the seventeen cases of meningitis, the spinal fluid was clear in four; the Nonne-Apelt test was

12. Di Vestea, D.: Apoplectic Contra-lateral Syndrome After Operation for Acute Meningitis on the Opposite Side, Rinasc. med. **2**:278, 1925.

13. Ohnacker, P.: Ztschr. f. Hals- Nasen- u. Ohrenh. **11**:350, 1925.

14. Karbowski, B.: Acta oto-Laryng. **7**:356, 1925.

positive; cytologically, there were from 15 to 20 mononuclear cells per cubic centimeter; these patients recovered. All the meningeal cases with turbid fluid, except one, died.

Yerger,[15] from seventeen personally observed cases of brain abscess, believes that the most important diagnostic method for the discovery of suspected intracranial pus collections is the examination of the cerebrospinal fluid. In his seventeen cases, the cell count was below 400 in 65 per cent. A complicating septic meningitis must be suspected when the count rises suddenly. Certainty is afforded only by the demonstration of micro-organisms. Of thirteen necropsies, 70 per cent showed meningitis.

In a later communication,[16] he reports eighteen cases which came to necropsy. He believes that early diagnosis can be made only on the basis of a combination of symptoms, not on a single change. Early spinal puncture usually reveals a "sympathetic meningitis."

(*b*) Papilledema: White's[17] contribution is based on 184 cases of intracranial complication of otogenic origin. He concludes that papilledema is a useful diagnostic sign in meningitis, brain abscess or infection of the lateral sinuses, but its absence does not exclude an intracranial complication. Three stages can be distinguished: pronounced papilledema, mild incipient papilledema, blurred contours or optic neuritis. Papilledema was noted in 38 per cent of the cases of cerebellar abscess, in 60 per cent of the cases of temporosphenoidal abscess, in 44 per cent of the cases of meningitis, and in 40 per cent of sinus infections.

In 50 per cent of the cases of meningitis with increased spinal pressure, the ocular fundi were normal. From the bacteriologic point of view exclusively, streptococci were found in the cases with papilledema. Increase of papilledema in the course of the disease is an unfavorable prognostic sign.

Lillie,[18] on the other hand, had four patients with choked disk, associated with mastoiditis, who recovered completely. From these he came to the conclusion that while there was no evidence of intracranial extension, it could not be definitely excluded. Three patients had parasinoidal abscess; the other a pachymeninigitis and a large cholesteatoma formation pressing on the sinus and cerebellum. One patient was subjected to intradural exploration, without result.

15. Yerger, C. F.: Left Temporal Lobe Abscess with a Report of Two Cases, Ann. Otol. Rhin. & Laryng. **33**:1364, 1925.

16. Yerger, C. F.: Case Histories of Otitic Brain Abscess Observed at Cook County Hospital, Ann. Otol. Rhin. & Laryng. **34**:258, 1925.

17. White, L. E.: Papilledema of Otitic Origin, Arch. Otolaryng. **2**:371 (Oct.) 1925.

18. Lillie, Harold I. and Walter I.: Choked Disc in Association with Surgical Mastoid Disease Without Apparent Intradural Involvement, Tr. Am. Otol. Soc. **17**:268-282, 1925.

Lillie believes that while the incidence of choked disks in surgical mastoiditis without intradural extension is low, it is sufficiently frequent to be considered in the differential diagnosis.

Hesse [19] asks: "Is the incidence of complications in acute inflammation of the middle ear dependent upon bacteriological or anatomical factors?"

He observed intradural complications in six of fifteen cases of infection from *Streptococcus hemolyticus;* nine of twenty-one from *Streptococcus nonhemolyticus;* seven of eight from *Streptococcus mucosus;* and one pneumococcus infection without complication.

(*c*) The Pulse in Suppurative Lesions: Lund [20] says that the pulse rate and spinal pressure in otogenic brain abscess may vary independently of each other. The slight increase of intracranial pressure in acute intracranial affections—such as meningitis—does not, as a rule, diminish the frequency of the pulse; but conditions differ with localized unilateral pressure, as, for example, in brain abscess and in acute cerebral hemorrhage. The latter is often associated with bradycardia, which is undoubtedly referable to the sudden rise of pressure. The opposite behavior—a sudden rise of pulse rate—is observed occasionally as an immediate sequel to opening a cerebellar or cerebral abscess. Lund thinks that localized pressure with unilateral action may be the most essential cause for the bradycardia in otogenic brain abscess, as his cases showed a marked bradycardia as often in abscess of the cerebrum as of the cerebellum, in spite of the close vicinity of cerebellar abscess to the vagus nucleus. The pressure of the abscess is not the only cause of the slow pulse; as it is relatively common in otogenic meningitis, in sepsis combined with pleocytosis in the cerebrospinal fluid, as well as in brain abscess and in acute otogenic encephalitis. Subdural abscess was associated with well marked bradycardia in one out of four cases.

Prognosis: Altogether, of 151 patients, 65 had a slow pulse, with a mortality of 81 per cent, whereas the mortality in 86 cases with a pulse proportionate to the temperature did not exceed 32 per cent; in other words, in acute otogenic intracranial complications, the mortality was high in cases of bradycardia. As Lund believes that the principal cause of slow pulse is apparently referable to a toxic irritation of the vagus nerve, the resulting bradycardia is therefore an unfavorable sign, and can be considered as analogous to the dangerous bradycardia (described by Traube) which is occasionally noted at the height of acute infectious diseases.

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19. Hesse, Walter: Arch. f. Ohren- Nasen- u. Kehlkopfh. **113** (June) 1925; abstr. J. Laryng. & Otol. **40**:675-676 (Oct.) 1925.

20. Lund, R.: Behavior of the Pulse in Labyrinthine Affections and in Otogenic Intracranial Complications, with Special Reference to Brain Abscess, Ugesk. f. Laeger 1926, no. 10, p. 246.

BRAIN ABSCESS

Relative Frequency of Suppurative Intracranial Complications.—Hirzinga [21] reviews the cases of brain abscess and purulent meningitis seen during the last fifteen years at the University of Groningen, Holland.

Of 28 cases of brain abscess, 6 originated as the result of acute otitis, and 22 from chronic otitis. The 28 cases of brain abscess included 4 of cerebellar abscess (one recovery); 24 of cerebral abscess (nine recoveries). In 14 cases, the correct diagnosis of brain abscess could be made prior to the operation. Contralateral paresis was present in 7, and aphasia in 5 cases. Of the ten patients cured, 2 had acute and 8 chronic cases.

Clinical Pathology.—The future advancement in the early diagnosis of local intracerebral collections of pus must depend on a better understanding of the blood supply of the different areas uniting the primary focus with the intradural contents. This can be obtained only by painstaking injection and corrosive cadavar work, such as found its first—as far as I know, its last—expression in the atlas of Adamkiewicz.[22] For it is now generally admitted that the vast majority of brain abscesses are adjacent and occupy rather definite sites in the temporal lobe or cerebellum, depending on the original focus in the ear or in the frontal lobe when from the nasal sinuses. However, when the pathologic process which causes the lesion is from a vascular embolus, the abscess may be situated far distant from the original focus.

Bleyl [23] reports an unusual "frontal lobe abscess as a sequel to otogenic sinus thrombosis," and calls attention to the fact that the latter may lead to the formation of brain abscess in one of three ways: (1) by continuity, (2) by the metastatic-embolic route (after passing through the pulmonary circulation), (3) by retrograde transportation of infectious material from the sinus into a meningeal vein. He believes that continuity should be considered especially in cerebellar abscess; that metastatic embolic abscess is usually situated in the marrow substance and the retrograde type in the cortical substance. He presents a case with intracranial manifestations two months after evacuation of the sinus and ligation of the jugular vein. The cerebellum and temporal lobe were explored without success. Necropsy revealed an extensive cortical abscess at the upper pole of the frontal lobe on the

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21. Hirzinga, E.: Intradural Complications of Acute and Chronic Otitis Media, Geneesk. Gids (Holland) **3**:561, 1925.

22. Adamkiewicz, A.: Die Blutgefässe des menschlichen Rückenmarkes, Sitz. d. k. Akad. d. Wissensch., Math.-naturw Cl. **84**:469-502, 1881; **85**:101-130, 1882. Die Arterien des Verlängerten Markes vom Uebergang bis zur Brücke, Vienna, 1890.

23. Bleyl, R.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:16, 1925.

same side, the cause of which was a thrombotic vein which passed from the abscess backward to the vein of the sylvian fissure and communicated with the transverse sinus through the anastomosing vein of Trolard.

Hofmann,[24] in an important communication, reports the result of a pathologic study of the course within the brain itself of six otogenic temporal lobe abscesses. He found that the pus takes a rather typical direction. When the abscess originates from a diseased point in the tegmen, it invariably has a uniform tendency to extend toward the inferior horn.

This is of great surgical importance and should lead to more exact exploration, in much the same way that studies of the limitation of pus by the fascia of the neck have improved the surgery of the upper cervical region.

His first case was a large abscess of ovoid shape, which extended forward almost to the anterior end of the lower horn and backward to the beginning of the posterior horn. Below, the abscess reached to the base of the brain. In its mesial direction, it communicated with the lower horn. However, the *rupture had remained localized through agglutination of the choroid plexus with the wall.* The abscess wall was formed by the greatly infiltrated, softened and hemorrhagic medullary substance. The portions around the abscess were edematous, the edema extending forward and upward to the upper temporal convolution.

The second temporal lobe abscess extended forward to near the temporal pole and backward into the occipital lobe beyond the posterior horn. The entire abscess was composed of two parts, the posterior portion being of simple structure, extending from the base, in a curve, toward the inferior horn and reaching close to it. The anterior portion consisted of several recesses; the largest, at the level of the middle temporal gyrus, extended forward nearly reaching the temporal pole. The abscess wall was formed by infiltrated medullary substance, which was very much changed. Locally, the abscess had invaded the cortex and in part destroyed it. There was no capsule formation.

In the third case there were two temporal lobe abscesses; one mesially situated, reaching close to the lower horn; the other lateral, much larger, reaching below to the base, while its upper end closely approached the mesial abscess, as if they were just about to unite. The abscess wall was formed by infiltrated, proliferating medullary tissue. The brain was extensively softened; the inflammation had locally involved the cortex and caused partial cortical atrophy. The ventricle contained pus; the choroid plexus was infiltrated and adherent to the wall at a place near the abscess.

His fourth case was a temporal lobe abscess, larger than a walnut, extending into the lateral ventricle.

A fifth brain showed a left sided temporal abscess, with circumscribed fibrinous internal pachymengitis in the immediate vicinity of the field of operation. There was a loss of substance in the course of purulent disintegration, which reached to the vicinity of the lower horn. There was also a small abscess in the upper convolution of the temporal lobe. This abscess was distinctly bounded anteriorly and above, passing gradually backward and downward into a region of inflamed, softened and necrotic medullary substance. The diseased portion comprised a large part of the temporal lobe and approached the inferior horn, the wall of which was likewise considerably involved, leading to the formation of pyocephalus.

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24. Hofmann, L.: Otogenic Temporal Lobe Abscesses, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:93, 1926. (From the Vienna University Clinic for Diseases of the Nose, Throat and Larynx.)

How a cortical abscess of the brain develops from a septic thrombophlebitis by nutritional death of the tissue and may thus remain sterile, while suppurative leptomeningtis is caused by a discharge of bacteria into the pial meshes, which leads to general bacterial leptomeningitis if the bacteria are not locally limited, is described microscopically by Turner and Reynolds [25] in a case of "Furuncle of the Right Nasal Vestibule: Septic Thrombosis of the Cavernous Blood Sinuses."

The brain presented a red area, 1 cm. in diameter, situated in the right frontal contiguous to the temporal lobe, on the one hand, and the caudate nucleus, on the other. Microscopic examination showed this to be an abscess. The brain was necrosed and infiltrated with cells, many of which were polymorphonuclear leukocytes. Liquefaction was just commencing in the center. The pus was continuous with that lying in the meshes of the sylvian fissure and along the pial sheaths of the larger blood vessels entering the brain in this area.

Relation of Brain Abscess to General Infections.—That brain abscess should at times be regarded from the point of view of a local manifestation of a general infection—as a metastatic embolic process—and not simply as an evidence of direct infection from a local focus in the ear or nose (although when such a condition coexists, it doubtlessly has some bearing on the etiology), is shown by the case of Siegmeister,[26] in which the route of the infection was apparently from a carbuncle in the right lumbar region, emboli from this area causing the temporosphenoidal abscess. This assumption was confirmed by the demonstration of staphylococci in pure culture in the pus of the brain abscess, as well as of the carbuncle.

Cases of Temporosphenoidal Abscess with Unusual Symptoms.—(*a*) Spontaneous Nystagmus: Tweedie [27] reports a case of cerebral abscess with spontaneous nystagmus.

A child entered the hospital with bilateral otorrhea of eight days' duration. Three days later the temperature and pulse were below normal, and bilateral optic neuritis appeared. Paralysis of the left external motor oculi supervened, with spontaneous nystagmus. Spinal puncture yielded a clear cerebrospinal fluid. There was frontal headache, without vomiting. Incision of the tense dura mater of the left temporosphenoidal lobe was followed by hernia of the brain. Exploration of the temporosphenoidal lobe liberated an ounce and a half of pus. The patient died. Necropsy was not performed.

The unusual appearance of the spontaneous nystagmus in a small temporosphenoidal lobe abscess calls for comment. In view of the absence of necropsy, the question must be asked, Was the nystagmus not a manifestation of an associated cerebellar abscess, especially as there

25. Turner, A. Logan; and Reynolds, F. Esmond: J. Laryng. & Otol. **41**:73, 1926.

26. Siegmeister, W.: Temporosphenoidal Abscess and Mastoiditis of Doubtful Origin, Laryngoscope **35**:787, 1925.

27. Tweedie: Otogenous Brain Abscess, Right Temporal Lobe with Normal Appearance of Tympanic Membrane, J. Laryngol. & Otol. **40**:467 (July) 1925.

was an accompanying (1) external ocular paralysis, (2) intense papilloedema, and (3) cerebral herniation? Statistics demonstrate that cerebral and cerebellar abscesses coexist in nearly 6 per cent of the cases, although up to the present not diagnosed during life.

(*b*) General Hyperesthesia, Except of the Arm of the Contralateral side: Carrasco [28] reports an otitic cerebral abscess, with hyperesthesia.

A man with a history of progressively severe headache followed by right sided ear suppuration of one month's duration, was deaf in the right ear. There was also a slight facial paresis and extreme hyperesthesia. No part of the head could be touched without producing extreme pain. This cutaneous hyperesthesia exended over the entire body. Light and noise likewise caused severe suffering. When the patient sat up in bed, the headache increased and nausea supervened. The pupils were narrow and rigid, the pulse slow (60); temperature was normal. On the seventh day after admission, he became more quiet, the somnolence being referable to morphin. *The cutaneous hyperesthesia persisted, except in the left arm, which was not very sensitive.* On the following day, the patient was in a state of deep coma, insensible to all stimuli. Mastoid exploration was negative. Two brain punctures for pus were without result. Necropsy showed softening of the right temporosphenoidal lobe, and an abscess containing greenish and offensive pus, without a capsule, the walls being formed by softened brain substance.

Cases of Intradural Involvement in which Accompanying Serous (Protective) Meningitis Contributed Outstanding Symptoms.—That localized arachnoiditis—protective or septic meningitis—is a frequent complication of extradural suppuration, I have ample clinical evidence. In several of my middle fossa cases it has produced hemianoptic indentations of the visual fields; and in one, a complete hemianopsia.

In the posterior fossa cases, I have had numerous examples of a complete disappearance of intense vertigo—dizziness, nystagmus—and papilledema after the evacuation of fluid from the subarachnoid meshes. Cisternal arachnoiditis simulating cerebellar tumor was first described by Horrick,[29] and, I think, is the explanation of Boenninghaus' [30] case of "Acute Serous Meningitis After Otitic Labyrinthitis with the Picture of Cerebellar Abscess; Cure by Incision of the Cerebellar Dura; Subsequent Development of a Meningocele in the Auditory Meatus, Probably from the Occluded Lateral Pontine Cistern."

The subsidence of the symptoms of a brain abscess following simple incision of the dura causes Boenninghaus to assume that there existed a serous extravasation in the ventricles. His explanation is that the incision of the dura allowed brain prolapse which led to diminution of the pressure. About a month after the incision a meningocele developed, which ruptured three times in the course of some months, but ultimately

28. Carrasco: Siglo méd. **75**:531, 1925.

29. Horrick: Arachnoiditis, Tr. A. M. A., Section of Nervous & Ment. Dis., 1923, p. 210.

30. Boenninghaus, G.: Ztschr. f. Hals- Nasen- u. Ohrenh. **13**:1, 1925.

closed of its own accord. During the entire period the patient felt perfectly well, which Boenninghaus thinks indicated that the meningocele was closed off from the remaining meninges. The origin of the meningocele is thus explained by Boenninghaus: The original serous meningitis produced an agglutination of the pia and arachnoid at the base of the skull, interrupting the communication with the fourth ventricle. A cyst formed in which cerebrospinal fluid accumulated, because the pontile cistern has a choroid plexus of its own. External influences (the first time by extraction of a sequestrum from the labyrinth) led to an increased secretion and rupture.

Sensory Aphasia from Extradural Abscess.—Thormann [31] reports a case of verbal amnesia and occasional paraphasia with an extradural abscess over the left temporal lobe, drainage of which was followed by recovery. The cerebrospinal fluid was always normal; consequently Thormann thinks temporal lobe abscess could be excluded. He asserts that verbal amnesia occurs often with extradural abscess in the left middle cranial fossa, but sensory aphasia rarely. The speech defect is explainable as a result of pressure in a large abscess; but in small ones by a collateral meningo-encephalitis. He thinks that his case teaches that sensory aphasia may occur with extradural otogenic abscess in the left middle cranial fossa, and consequently cannot be regarded as pathognomonic of temporal lobe abscess.

In reviewing Thormann's case, it might be suggested that (1) an incapsulated abscess still exists—a far from unknown condition—to which Ott also calls attention when he says: "Even though the symptoms of intracranial infection subside spontaneously, these patients should be regarded as abscess suspects, especially so if an occasional attack of headache, vertigo and loss of weight persist." [32]

More probably, however, the aphasia in Thormann's case was the result of (2) an incapsulated pia-arachnoid accumulation of cerebrospinal fluid over the temporal lobe, which was relieved by the evacuation of the extradural abscess.

The motor cortical area for speech is susceptible to slight and transient pressure, such as pial hemorrhages, but sensory aphasia without an intradural complication is almost unknown. It is, however, a frequent occurrence in localized cortical pia-arachnoid collections of pus. In Thormann's case, I would suggest that the disappearance of the aphasia after the evacuation of the extradural abscess is best explainable by the supposition that the accumulation of protective intradural fluid near the extradural collection was not infected, or but slightly so.

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31. Thormann, H.: Sensory Aphasia with Otogenic Extradural Abscess in the Left Middle Cranial Fossa, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:429, 1925.

32. Ott, W. O.: Intracranial Complications of Ear and Accessory Nasal Sinus Infections, Texas State J. Med. **21**:12, 1925.

MacCuen Smith,[33] in reporting "Three Cases of Otitic Brain Abscess," shows that in all these cases there was almost entire absence of neurologic and ophthalmologic symptoms, while in one of the cases the probability is that the patient had a brain abscess for three years before he came under Dr. Smith's care. The abscess did not seriously interfere with the patient's work until a few weeks before he was admitted to the hospital.

33. MacCuen, Smith S.: Ann. Otol. Rhin. & Laryng. **34**:109, 1925.

(To be continued)

Abstracts from Current Literature

Ear

Concerning Disease Symptoms in the Region of the Otolith Apparatus.

Thielemann, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:557 (March 20) 1926.

According to de Kleyn and Versteegh, cases of otolith disease in which tonic neck reflexes could be excluded with certainty have not yet been described, but they do occur, according to Nylen. Thielemann believes that he observed a patient with pure tonic labyrinth reflexes. A man, aged 49, could not lie down in bed for two weeks. As soon as he lay on his back, he said, he became dizzy and nauseated. He was obliged to sleep in a sitting position. He had never suffered from the ears, although he did not hear so well lately. Up to this time he had no dizzy spells. He denied any more serious disease or infection. The otoscopic examination showed normal conditions. The functional test showed a bilateral hardness of hearing attributable to the inner ear, which was more pronounced on the left side.

When the patient stood upright, there were no spontaneous nystagmus or past pointing. Turning nystagmus to the left lasted twenty-two seconds and past pointing took place in both arms outward. The turning nystagmus to the right lasted thirty-four seconds. Both arms past pointed outward. Caloric minimal irritation produced nystagmus on both sides, but only the corresponding arm past pointed. When the patient was lying on his back, a weak, slightly rotatory spontaneous nystagmus appeared to the left. When the head has slowly turned to the left, with the patient in the same position, nystagmus increased, and reached its maximum when he was lying on his left side. The patient then became nauseated. When the head was slowly turned to the original position the nystagmus decreased. When the patient's head was turned from 20 to 30 degrees to the right, the nystagmus ceased. The patient could keep this position without becoming dizzy. If the head was turned further to the right, a slight rotatory nystagmus was noticed, but this time to the right. The intensity increased the nearer the head approached the right lateral position. In the ventral position, a slight nystagmus to the left was noticed. When the left labyrinth was brought downward, namely, when the head was turned to the right, the nystagmus to the left increased and became the strongest with the head in the lateral position. If in the ventral position the head was turned to the left and the right labyrinth faced downward, a nystagmus to the right occurred, although of less intensity. If in the upright position, the patient's head was bent to the left shoulder, plain, slightly rotatory excursions of the bulb occurred to the left. Corresponding excursions to the right were noticed when the head was bent to the right shoulder. With the head hanging, no nystagmus could be shown. Consequently a rotatory nystagmus occurred as soon as a labyrinth was brought into a lateral position from any other position. The excursions were the plainest when one labyrinth was placed under the other. The nystagmus has always been directed toward the labyrinth occupying the lowest position. In the dorsal position, a position of rest could be found when the head was turned from 20 to 30 degrees to the right. In order to exclude the tonic neck reflexes the head was fixed into relation to the body. When the patient in the dorsal position was turned to the left or right, the same results were obtained as described before. The

same occurred in the ventral and in the upright position. The isolated tonic neck reflexes were then examined. While the patient was in a dorsal position, the head was fixed with the hands so that it was turned at an angle of 25 degrees toward the body. In this position no nystagmus was present. If the trunk alone was turned to the right or to the left, no nystagmus occurred. When the body was bent to the right or to the left, in a sitting position, no nystagmus occurred when the head was always kept in an upright position; when the patient was lying on either side nystagmus was observed. When the head was then lifted so that it stood upright, namely, if in a lateral position of the patient the head was at a right angle to the right or left shoulder, the nystagmus and the feeling of dizziness ceased. The nystagmus persisted so long as the head remained in the corresponding position in space. It could be observed for two or three minutes. Nausea prevented further observation. If the left labyrinth was lying below, nausea occurred in a short time. Past pointing was plain in the dorsal position when the head was in a lateral position. It was always directed to the side opposite to the nystagmus. With this vasolabile patient, the morbid otolith reaction was stronger to the left than to the right side. This patient also suffered from urticaria factitia. The neurologic examination showed a slight caput quadratum and teeth hypoplasias, which point to an early disturbance of the epithelial bodies, and a minimal exophthalmos. The right pupil was slightly narrower, and the right side of the face was a little more reddened than the left. There was a scar from a previous injury behind the right ear which may have caused a slight disturbance of the sympathicus. For a year, a decrease of sexual potency and a diminished power of memory were noticed. The picture was that of inner secretive, vegetative nervous disturbances.

Thielemann thinks that the patient presented a picture of an otolith disease with purely labyrinthian tonic reflexes. Borries claims that it has not been proved that the otoliths produce, or do not produce, nystagmus in man. The otoliths and the proprioceptors of the neck furnish the legitimate position of rest of the eyes in reference to space and to dependence of the head to the body. In man with prevailing optic orientation, this mechanism of regulation loses its importance, and yet the otoliths are one of the many sources of the tonus for the muscles of the eyes, although they are not the most important source. There can scarcely be a doubt that the otoliths can produce nystagmus, under pathologic conditions. Brunner remarks that so far labyrinthian symptoms came to our attention which were elicited by the semicircular canals. This is explained by the fact that reflexes from the semicircular canals are only slowly compensated, whereas symptoms on the part of the otolith apparatus are quickly compensated, as a rule, and they become manifest only if the compensation is suppressed for some reason or the other, or if the disturbance of innervation is fixed. So far, a static attack of nystagmus elicited by the otolith apparatus has been assumed. Thielemann thinks that a more definite diagnosis can be made in the region of the otolith system. The Utrecht school has informed us concerning the function and position of the otolith apparatus; also for man, the main portions of the utriculus and sacculus are the principal functional spots of the statolith system. When the head is in the normal upright position, both main portions of the utriculus are located so that the otoliths press on the membrane. This, according to Magnus, is the so-called minimum position, the position of rest. When the head points downward, the otoliths of the utriculus hang down from the membrane; this is the maximum position. Any other position of the head increases or

diminishes the always present tonus of the utriculus. The main parts of the sacculus are differently arranged. Every main part of the sacculus stands always at an angle of about 90 degrees to the main part of the utriculus. Although both main parts of the utriculus lie in a horizontal plane when the head is upright, the main parts of the sacculus are inclined so that the imaginary lines of continuation converge upward at a very acute angle. The continuation lines of the main parts of the sacculus also converge backward at an angle of 50 degrees (de Burlet). Each main part of the sacculus forms dorsally an angle of about 25 degrees. The maximum position of irritation of the otolith of the sacculus corresponds to the hanging otoliths; the position of rest to the pressing otoliths. The otoliths of the main portion of the left sacculus hang down when the head is in the left lateral position. At the same time the otoliths of the right sacculus are in the minimum position. The tonus of the main parts of the sacculus can only be at rest if each main part of the sacculus stands at the same angle to the vertical line. For instance, bending of the head to the left irritates the left main portion of the sacculus more and more whereas the right main portion of the sacculus approaches more the position of rest. Thielemann's patient showed that the disturbance of the right sacculus otolith system was not of the same intensity as on the left side. This overponderance on the left side was the cause, in a dorsal position, that the compensatory sacculus otolith reaction had not been reached when the head was in a symmetrical position to the median line of the body, but only when the left main portion of the sacculus was perpendicular to the horizontal plane, had the left sacculus otolith irritation reached exactly the line of maximum and minimum position. This case seems to furnish the first clinical proof for the position of the otoliths in man, as de Burlet has figured them out in his histologic specimens. The work of German is also explained. German observed a patient in whom symptoms showed that not only a certain definite position of the head, but that a position at any point of an arc, respectively, of a spherical segment caused dizziness.

A uniform etiology could not be found in the cases of otolith disturbance. Grip, toxic causes, inflammatory processes in the middle and inner ear and syphilis have been mentioned. Borries claims that injury to the head can cause otolith disease. Brunner observed that, in dull injuries to the head, nystagmus with dizziness occurred in the lateral position of the patient, which was of little intensity in the dorsal position. Brunner thinks of the possibility of diseased vestibular sacs because in animal experimentation he frequently found a copious exudate, especially in the cisterna perilymphatica. In one case he saw these attacks of nystagmus toward the healthy side at the time when the semicircular canals did not react to caloric irritation any further.

The static apparatus can be influenced by way of the vasomotor tracts by injuries which attack the vegetative system. A number of authors are quoted. Sometimes the primary cause may lie in the central nervous system. Therapeutically, Thielemann administered barbital in small dosages, 0.1 Gm. from two to three times daily. In order to influence the vessel system he gave 0.0001 Gm. of atropine from two to three times a day. In his patients the attacks diminished after a short time, three weeks at the most. The two first of his four patients reacted after a few days. Thielemann emphasizes the statement made by Voss, who claims that an examination of attacks of dizziness is only complete if the otolith apparatus, as well as the semicircular canals, are examined.

ALCOHOL DOUCHES WITH THE TYMPANIC TUBE IN CHOLESTEATOMA. H. THORMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:573 (March 20) 1926.

Thormann states that, for decades, the syringing of the epitympanic cavity with pure alcohol is used in Rostock in order to remove cholesteatomatous masses and to dry the cavity. Thormann always uses water first and then alcohol in small quantities, from 8 to 12 cc. These small quantities do not excite the labyrinth. Thormann claims that the technic used in Rostock is more simple than the technic of Hopmann.

AMBERG, Detroit.

FROM THE PROCEEDINGS OF THE GERMAN OTOLARYNGOLOGICAL SOCIETY

THE ROENTGEN-RAY PICTURE OF THE MASTOID AND ITS CLINICAL INTERPRETATION. STEURER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:504 (supplement) (Nov.) 1925.

Three hundred temporal bones of nondiseased subjects, decalcified and sectioned, showed marked agreement of the findings with the previous readings of distribution of the cells, abnormal cell structure, position of the sinus, course of the emissary, etc.

In some, deep lying cells which were overlayed by compact bone escaped detection. Veiling of well developed cells sometimes occurred through a thick cortex. Such pictures may mislead.

In Tubingen, in the last two years, 1,400 pictures were made for diagnostic and prognostic purposes. The pictures were especially valuable in the masked cases of *Streptococcus mucosus* and pneumococcus infection by revealing cell destruction when clinical symptoms were scant.

Orientation is easier if a picture is taken early in the disease for comparison with later ones; failing in this, the normal side may be taken for comparison. In 737 patients, 527 showed identical structure on both sides. Irregular or imperfect pneumatization and thick walls may cause great difficulty in interpretation. Cholesteatoma may be diagnosed and outlined, and erosions of the sulcus toward the middle fossa may be diagnosed. In forty-two cholesteatoma cases, the extent was shown in thirty-one.

Many illustrations were shown of the various conditions described.

A NEW PRINCIPLE FOR AIDING THE HEARING. PERWITZCHKY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:593 (supplement) (Nov.) 1925.

The speaking tube and the telephone and microphone apparatus have their limitations, the former because of its inconvenience and distortion of tones by its own fundamental tone, the latter because current strength cannot be transformed into electromagnetic force so as to operate the telephone diaphragm proportionately. Most cases of deafness affect principally either the lower or higher ranges of tone. The author suggests that since the development of the radio has shown the possibility of transforming frequency of sound waves so as to raise or lower tones an octave, at least for pure tone, it may be possible to work out the same principle for the mixed tones of speech. He says that experiments in this direction are so promising that an electrotechnical laboratory is now working on it.

ROENTGEN-RAY TREATMENT OF THE TUBA AUDITIVA. AMERSBACH and WUCHERPFENNIG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:511 (supplement) (Nov.) 1925.

The rays are applied through the mouth by a special technic evolved after careful anatomic measurements. At first, only patients in whom a radical operation was performed with persistent discharge were treated, but now these authors are also treating chronic suppurative otitis media.

LEWY, Chicago.

Larynx

A CASE OF ASPIRATION OF A TOOTH PROSTHESIS INTO THE LARYNX. S. KOMPANEJETZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:484 (Jan. 18) 1926.

Up to the year 1916, about 2,000 cases of foreign bodies in the larynx, trachea and bronchi have been described, according to O. Chiari. Kompanejetz reports the case of a patient who complained that he had swallowed a prosthesis with four teeth while he was asleep. Roentgenoscopy showed a foreign body, the lower end of which was visible 2 cm. above the jugulum sterni. In the surgical clinic the esophagotomia externa was performed. The prosthesis could be palpated in the esophagus. A bougie was introduced by the assistant into the esophagus through the mouth for better orientation. The patient vomited and became very cyanotic. An emergency tracheotomy was necessary. The author detected the foreign body in the larynx and removed it by laryngofissure under chloroform narcosis. In spite of the fact that the foreign body had remained twenty-eight days in the larynx, decubitus was not present, but the motility of the vocal cords was interfered with, probably on account of a perichondritis.

THE PHYSIOLOGY OF THE RECURRENT NERVE. F. V. D. HUETTEN, Ztschrf. f. Hals-, Nasen- u. Ohrenh. **13**:601 (March 20) 1926.

V. d. Huetten previously recommended the position at rest of the diseased half of the larynx in the treatment of tuberculosis of the larynx by icing the surgically exposed recurrent nerve. He reported six cases with favorable results. It was observed that the blocking of the recurrent nerve caused disturbances. In five cases the labium vocale became immovable in an intermedial position immediately after icing, which position occasionally approached the median line, but which left a plain triangular slit during phonation. The arytenoid cartilage was inclined, more or less, forward in four cases, and the labium vocale was excavated in three cases. In four cases, the mobility of the arytenoid cartilage could be noticed in spite of interruption of the recurrent nerve and paralysis of the corresponding labium vocale.

According to Leichsenring, the fibers of the transverse or oblique muscles are provided for by the superior laryngeal nerve, or by the recurrent nerve of the other side. In one patient, Leichsenring found an anastomosis of the recurrent nerve with the superior laryngeal nerve, and in another he found this mobility of the arytenoid cartilage in spite of interruption of the superior laryngeal and recurrent nerves of the same side. Some authors state that the recurrent nerve carries sensory fibers. V. d. Huetten made this circumstance the subject of investigation, and came to the conclusion that a decrease of sensibility on the operated side (touch, pain, temperature) could not be established. Yet he admits that patients with local affections of the larynx are not fit subjects for examination. The interesting investigations of Leichsenring, who blocks the recurrent nerve in normal larynxes with procaine solution,

are free from such errors, if the procaine solution affects only the recurrent nerve and not other sensory paths by diffusion. The author irritated the cocainized larynx with the galvanic current (3 to 5 milliamperes). The vocal cord, which was placed at rest, did not react; the other did. V. d. Huetten states that the blocking of the recurrent nerve furnishes many problems.

AMBERG, Detroit.

Nose

PAPILLITIS, HEADACHE AND MENTAL DEPRESSION FROM OSTEOMA OF SEPTUM. OTIS WOLFE, Am. J. Ophth. **9**:340-341 (May) 1926.

Wolfe reports a case of a man, aged 48, who, thirteen years previously, had been kicked in the head by a horse, following which he began to have headaches. These seemed to start in the occipital region and then would involve the whole head. The patient was never entirely free from headache and pressure in his head, but at times had attacks lasting for two or three weeks. In the last two years the headaches were so severe that he was completely incapacitated. The pain was most severe between 10:00 a. m. and 2:00 p. m., and was so severe as to require opiates.

The patient was referred to the author on account of a feeling of pressure back of the right eyeball, and the blurring of vision in this eye. At this time he was in a hospital suffering from an acute attack, and opiates were necessary to quiet him. A roentgenogram revealed fracture of the inner plate of the skull in the occipital region, approximately where he had been kicked. Operative measures on the skull to relieve the intracranial pressure were being considered.

The vision in the right eye was 20/30. It was not improved by refraction. The right disk margin was fuzzy and slightly edematous, with engorgement of the veins. Perimeter findings were suggestive of a scotoma with slight enlargement of the blind spot. With the campimeter, a small, but positive, central scotoma was outlined for red and blue.

Nasal examination showed only slight obstruction to breathing. The septum was deviated to the right. There was a large compensatory enlargement of the left middle turbinate. The septum did not shrink much with epinephrine and cocaine, and a cotton tipped probe could not be passed between the right middle turbinate and the bulge of the septum. The roentgenogram revealed a very thick, bony septum. At this time it was elicited that the patient's symptoms were precipitated and exaggerated if he caught the least cold in his head.

Surgery on the occipital region was deferred until the nose could be operated on, and under local anesthesia a submucous resection was performed. Bending sharply to the right and extending out from the sphenoid rostrum, the posterior septum was solid bone and varied from 5 to 7 mm. in thickness. It was tightly wedged up against the right middle turbinate. The sphenoid was exposed, but no pus or necrosis was found.

A few hours after leaving the operating room the "pressure feeling" seemed relieved. The next day he had very little headache, and the change in his mental condition was very noticeable. From the third day on, he had absolutely no headache and has had none since. The patient has become entirely different, and his mental condition has completely cleared up. A few months after the nasal operation, vision was 20/20 and no scotoma was found.

The author feels that obscure eye symptoms, what he calls sunpain (i. e., pain between 10:00 a. m. and 2:00 p. m.), and headaches call for critical consideration of the posterior nose even though no pus is present. If the patient's symptoms are exaggerated by catching cold, it is very significant.

RETROBULBAR NEURITIS DUE TO FRONTAL SINUSITIS. L. WALLER DEICHLER, Am. J. Ophth. **9**:346-348 (May) 1926.

Deichler reports a case of retrobulbar optic neuritis with the history that for the past four days there was noticed increasing difficulty in reading with his left eye and an increasing fog in front of both eyes. His vision in the right eye with glasses was 15/15, in the left eye 15/200. The disk margins were well defined; the nerve of good color; the vessels and the tension were normal. The visual fields showed a marked contraction of the form fields in both eyes; color fields were especially contracted in the left eye. There was an absolute central scotoma for red and relative scotoma for green in the field for the left eye, and a large relative scotoma for green in the field for the right eye.

After four days of elimination treatment, in the left eye in the macula and just below, two small round hemorrhages the size of a pinhead were noted.

Roentgenograms of the sinuses were negative; the sella turcica was negative but two abscessed teeth were found. These were extracted a few days later. On the day of extraction the macular spot had undergone slight change, marked by a deep red ring of congestion with a yellow center. The fields showed progressive contraction, and two weeks later there was marked contracture with almost complete loss of the nasal field in the left eye both for form and color. Vision had improved to 15/70, however.

The next day the disk had become somewhat fluffy with beginning edema, and the choroidal disturbance to the temporal side of the disk was more marked. There was now some incoordination; the station was somewhat disturbed and reflexes were exaggerated on the right side.

The following day a history of an acute sinusitis on the left side occurring two months previously was obtained. The discharge had suddenly ceased three or four days before the eye began to bother the patient.

The patient was referred to a rhinologist when a diagnosis of left ethmoiditis was made. An operation was performed with removal of the middle turbinate and opening of the ethmoid and sphenoid cells. No pus was found. No changes were noted with the ophthalmoscope until five days after operation, when the deeper layers of the nerve began to show pallor, yet the vision had improved to 15/50. There was, in addition, a low grade of neuroretinitis. Nine days after the operation his vision improved to 15/40, at which time he began to complain of a pain over the left frontal region which increased in severity. Ten days later the vision showed reduction and the ophthalmoscopic examination revealed arteries which seemed to be contracted in comparison with those of the right eye. A few days later the left frontal sinus was operated on by the nasal route. Much pus was found and freely evacuated. The ophthalmoscopic examination revealed increasing paleness in the deeper layers of the nerve. The fields showed a spreading, especially to the nasal side, both to the form and color field.

Because the pain persisted and the sinus condition was unsatisfactory, a modified Killian operation was performed two months after the patient was first seen. Pus, degenerate membranes and polypoid growths were found. A week after operation his vision had improved to 15/15. While the optic

nerve was white, it seemed to be improved in color and was certainly improved in function. The color sense, which had been much interfered with from the beginning, was now improved.

When last seen, the patient had 15/15 vision in the left eye and 15/10 in the right.

H. H. VAIL, Cincinnati.

A CHONDROMA OF THE ETHMOID WITH OPERATION AND CURE. E. SCHLITTER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:489 (March 20) 1926.

So far, fifty-two cases of enchondroma of the nose have been described; Schlitter also describes a case. The incision in the skin and soft parts was done according to Moure. The lamina papyracea was laid bare, and the ethmoid was opened. The largest portion of the right middle turbinate was removed. The tumor was hanging down with a thin stem from the nasal plane of a middle ethmoid cell, so that it could be removed easily with the scissors.

THE FATE OF REIMPLANTED CARTILAGE OF THE SEPTUM. DESIDERIUS V. KASSAY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:617 (March 20) 1926.

In the reimplanted cartilage of the septum, signs of beginning reconstruction appeared on the twelfth day. The vessels sprouted, cartilaginous cells proliferated, substitution by connective tissue took place and the matrix showed local and fibrous changes. The regressive changes consisted of separation of sequestrums, decalcification of the marginal zone and pyknotic disintegration of the nuclei.

AMBERG, Detroit.

Miscellaneous

ADENOIDISM AND ABNORMALITIES IN THE CONFORMATION OF THE NASAL SEPTUM AND THE UPPER JAW. PASQUALE RUSSI, Supplement to Arch. ital d. otol., rinol. e laringol. **2**:1-79, 1925.

In subjects with adenoids, abnormalities in the conformation of the bones of the face are: 1. Malformations (deflections, spurs or spines of the nasal septum, and, in the upper jaw, an ogival, often asymmetric, palate) nearly always due to general causes that affect the regular development of the fetus (syphilis, rickets). Malformations of this class range in gravity all the way from insignificant manifestations to the various kinds of harelip and cleft palate. 2. Malformations due to disturbances manifested in extra-uterine life and resulting either from general causes (lymphatism, scrofulosis) or from local causes, among which adenoid growths play the principal part. Malformations of this class may affect only the septum and the upper jaw but not necessarily both. While ogival palate may not be invoked as the sole cause of deflections of the septum, it is without doubt the principal factor. Deflections of the septum, ogival palate and abnormal arrangement of the teeth (dental ataxia) were found associated in 9 per cent of the cases studied. Ogival palate was associated with deviations of the septum in 43 per cent of the cases. Adenoidectomy restores completely nasal respiration impaired merely by the hyperplastic pharyngeal tonsil. But in subjects who present deflections of the septum or marked thickening or hypertrophy of the turbinates the same benefit is not produced. Sometimes, several months after adenoidectomy, beneficial modifications in the ogival palatine arch and in the dental arches may be observed.

WOLCOTT, Chicago.

ESOPHAGOSCOPY AND GASTROSCOPY. (A REMARK ON THE ARTICLE OF W. STERNBERG, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:138-146). FRITZ SPECHT, ibid., p. 592 (March 20) 1926.

Specht takes issue with some idea of Sternberg. The kneepieces, placed at an angle which Sternberg recommends, appear to Spohn to be extraordinarily dangerous. Specht claims that two patients, on whom Sternberg himself employed the instrument, suffered from the instrumentation. One patient collapsed and died. Necropsy revealed an extensive tearing of the esophagus. The other patient developed a retro-esophageal phlegmon and mediastinitis. A tube similar to Sternberg's should not be introduced into the esophagus, although excellent pictures can be obtained of the ballooned wall of the stomach, as Sternberg has demonstrated in other patients.

OF WHAT SIGNIFICANCE IS THE BLOOD PICTURE IN OTOGENOUS DISEASES? PRIZE ESSAY, BERLIN MEDICAL FACULTY. ELSE LEVY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:495 (March 20) 1926.

Else Levy furnishes an exhaustive essay on this subject. Two main groups must be differentiated concerning the changes of the cell elements in the blood: (1) Those which are caused by a disease of the blood forming or the blood destroying organs; (2) those which are caused by a disease of other organs or systems of organs which cause secondarily an irritation of the very sensitive hematopoietic apparatus. There are many individual and physiologic variations. Different persons react differently to the equally localized, equally constituted and equally graduated irritation. Levy's material was taken from the lobe of the ear, in the forenoon, before the patients had dinner.

The eosinophils are of great importance. There exists a congenital constitutional eosinophilia, but it is very rare. If an increase of eosinophils is found, a worm disease must be thought of; irritation of the vagus should also be considered, as it is also frequently found in diseases of the skin. Of the infectious diseases, scarlet fever shows an eosinophilia contrary to other diseases, even if the exanthema is missing. It is also found in gonorrhea. In septic infections eosinophils are frequently missing. The eosinophils are an important indicator of the gravity, respectively, of the course of such an infection. If they decrease or disappear entirely, a grave general disturbance may be assumed. If they reappear and if their number increases more than normally, a discontinuation of the injurious influences can be assumed, be it by remission of the suppuration or by encapsulation ("dawn of recovery," Schilling). It is not sufficient to establish the total number of neutrophils. The condition of the single cells must be considered. Arneth has given an exact classification of the neutrophils according to the number of nuclear segments, and he has determined hereby the age of the single cells. Pappenheim, Brugsch, Schilling and others have shown that the number of nuclear segments is not indicative of the age of the cell. Schilling's hemogram furnishes a clearer basis for judgment of the processes in blood formation, respectively, blood flooding by laying stress on the chromatin conditions of the nucleus and the quality of the protoplasm. Normally, only from 3 to 5 per cent of neutrophils reach the bloodstream with an unsegmented nucleus (rod nucleated); the other 58 to 66 per cent are segmented. In suppurative diseases, not only an absolute increase of neutrophil cells takes place but also the so-called youth forms are found. These are cells with an undivided nucleus, which can be stained less intensively, and frequently with plain nuclear corpuscles. In very severe irritation of the

bonemarrow, myelocytes are frequently found in the blood stream cells with bluish protoplasm, fine granulation and a round nucleus. According to the diagram a more or less pronounced shifting to the left is spoken of, if the myelocytes are present. This shifting to the left indicates a turn for the worse or a continuation. Its disappearance indicates an improvement of the disease process. The appearance of the young forms with the mostly simultaneous presence of neutrophilia is considered a defense reaction of the body, and Schilling calls this stage the "neutrophil phase of the fight." In this stage, with the continuous presence of nuclear shifting, the decrease in the number of leukocytes is prognostically very unfavorable.

Levy comes to the following conclusions: It is impossible to recognize the localization of a disease focus, but, in otogenous diseases, conclusions can be made with probability to a degree. Darling, Faltas Urbantschitsch and others have shown, and Levy has confirmed it, that a brain abscess usually produces a less pronounced change of the blood picture than sinus thrombosis, sepsis or erysipelas, which show similar blood pictures, but which show a less pronounced change than meningitis. Concerning the differential diagnosis between otitis externa and mastoiditis, it is stated that leukocytosis neutrophilia and more pronounced shifting to the left suggests mastoiditis. If the blood picture is only little changed, it cannot be excluded absolutely. These comparisons show that the blood picture can be an important symptom in infectious diseases showing the reaction of the body, but it is only one symptom. It may warn that the process is progressing, but in otogenous diseases the foci may be so small that the blood picture is little or not at all influenced, especially when the danger exists that the disease may progress into the sinus or into the meninges. The processes are small; therefore, great care must be taken that a normal or little changed blood picture may not deter one if an interference is clinically indicated. Furthermore, a single examination makes the interpretation of a blood picture more difficult, because no comparison is available concerning the individual reaction of the patient. Nevertheless, Levy could show that frequently a connection exists between the gravity and the course of the disease and the blood picture. The simple leukocyte count is not sufficient. The percentage of the various kinds of leukocytes must be observed and their rise and fall, disappearance and reappearance, if a diagnosis judgment concerning the gravity and the course of the disease is to be obtained.

The presence of youth formations must be emphasized, because not only the neutrophilia is significant for the degree of irritation of the bone marrow but the flooding by more or less unripe forms. The disappearance of eosinophils and the decrease in lymphocytes point to a more unfavorable prognosis, or to the appearance of a complication. Their reappearance and their increase is a sign of restitution beginning and is prognostically favorable. Some infections, for instance, scarlet fever, have a characteristic blood picture. This makes it possible to isolate the patient before the clinical symptoms are clear. If the hematopoietic apparatus is the primary focus of the disease and the otitis only a local manifestation, then the blood picture furnishes an explanation for the clinical symptoms. Therefore, the morphologic picture of the blood should be studied in cases of inner ear deafness, especially in those with Méniere's syndrome. In a number of cases of otogenous diseases, the blood picture can indicate the localization of the process, the prognosis and the complications. In cases of unknown etiology the blood picture may point to the cause of the disease, or it may prove it directly. Levy cannot form a final judgment concerning the value of the determination of the sinking rapidity

of blood corpuscles compared with the blood picture. She is unable to decide whether the determination of the sinking rapidity shows the progress of a suppurative process more quickly, because her material was not large enough. It is certain that the sinking reaction improves more slowly than the blood picture. In chronic middle ear suppuration it does not appear to indicate an operation, yet the sedimentation test is recommended in grave cases.

AMBERG, Detroit.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

March 17, 1926

FIELDING O. LEWIS, M.D., *in the chair*

SOME PHASES OF STREPTOCOCCIC INFECTION IN ACUTE MASTOIDITIS. Presented by DR. RICHMOND MCKINNEY, Memphis (by invitation).

Dr. McKinney, on account of recent experiences with mastoid cases, has come to believe that the type of infection plays a much greater rôle in the prognosis and clinical symptoms than he formerly thought. Three patients on whom he recently operated were cited. All were children with symptoms of acute mastoiditis demanding operation. Each showed a hemolytic streptococcus in pure culture. One showed positive clinical evidence of partial sigmoid sinus thrombosis, with a blood culture positive for the same organism. Operation was postponed on account of the opinion of the bacteriologist, Mr. L. O. Dutton. Mr. Dutton claims to be able to foretell the results of streptococcic blood stream infection with 100 per cent accuracy. He recognizes several types of streptococci existing as mixed strains, each of which is combined with a bacteriophage. When grown on artificial mediums, this balance is disturbed and one or the other predominates. Conclusions are drawn from the manner of colony growth and the morphologic characteristics of the organism. He has observed cases in which the bacteriophage predominated to clear up from a blood stream infection in seventy-two hours. When the organism predominated, the blood culture remained positive until a fatal termination.

DISCUSSION

DR. GEORGE M. COATES said that to him the prognosis value of the manner of growth of cultures obtained from mastoid wounds and the blood stream was a new idea. If this factor is constant in a large group of cases, it will be of undoubted value in the estimation of the proper time for surgical interference. The organisms of epidemics have, in his experience, shown a tendency to be more virulent than the sporadic cases. In a series of hospital army cases, Dr. Coates found that pneumonococcic mastoiditis was much more difficult to control than the streptococcic variety. In civil practice, many streptococcic cases are fulminant but show a tendency toward self limitation.

DR. GEORGE WOOD considered epidemic ear complications to be less severe than the sporadic cases.

DR. JAMES A. BABBITT stated that his tendency is to show more conservatism toward ligation of the jugular vein and interference with the sinus in thrombosis cases. He considered it most important that it be definitely established when and when not to operate in these cases. The question of the value of intravenous dye therapy was raised.

DR. FIELDING O. LEWIS asked how many cases had been observed in the manner described.

DR. MCKINNEY stated that Dr. Dutton has reported a series of ten cases of sepsis in which the prognosis was accurately made by the nature of organism growth. He, too, has a greater tendency toward conservatism when blood stream infection exists. In some cases he has used intravenous dye therapy with questionable results.

ANEURYSM OF THE AORTA. Presented by DRS. FIELDING O. LEWIS and C. J. BUCHER (by invitation).

Dr. Lewis was called to perform a tracheotomy on an adult male on account of dyspnea. On examination, the classical signs of laryngeal obstruction were absent, and the patient was removed to the hospital for study. Soon after his arrival, the dyspnea became so marked that it was necessary to give relief if possible. Previous blood studies reported a slightly positive Wassermann reaction and roentgen-ray examination showed no aneurysm. Dr. Lewis Clerf was called in to introduce a bronchoscope. He was unable to enter the left bronchus, and entered the right with difficulty. When this was done, the patient breathed more easily. A Jackson cane shaped cannula was then inserted, giving the patient some relief from dyspnea. The appearance was that of mediastinal neoplasm or new growth in the lung. A Wassermann reaction made later was strongly positive, and roentgen-ray studies by Dr. Manges showed a definite aortic aneurysm. The patient died from asyphyxia in a few days. Dr. Bucher demonstrated the specimen.

SERIAL FROZEN SECTIONS OF THE THORAX IN STERNUM-ERODING ANEURYSM OF THE ARCH OF THE AORTA. Presented by DRS. GEORGE W. NORRIS and GEORGE FETTEROLF.

Apropos of the case reported by Dr. Lewis, Dr. Fetterolf demonstrated slides of two cases previously observed and reported by Dr. Norris and himself. Soon after death the bodies were treated with formaldehyde, frozen and massive sections were made through the thorax. These sections definitely show the possibilities of pressure that may be exerted by aortic aneurysms. Full accounts of these cases may be found in the *Annals of Otology, Rhinology and Laryngology*, September, 1917, and in the *Archives of Internal Medicine*, July, 1920.

DISCUSSION

DR. LOUIS CLERF stated that it is unwise to do endoscopic procedures in cases of aortic aneurysm. Here the emergency demanded action, and previous observers had ruled out aneurysm.

CLOSURE OF RETRO-AURICULAR FISTULA. Presented by DR. JAMES A. BABBITT.

Dr. Babbit presented a girl, aged 14, on whom he had performed a plastic procedure for the closure of a retro-auricular fistula of six years' duration. The opening was one-third inch in diameter and had persisted after mastoid operation. He used a modified Troutman procedure with excellent cosmetic results.

April 21, 1926

GEORGE FETTEROLF, M.D., *in the Chair*

APHONIA. A REPORT OF THREE CASES. Presented by DR. WILLIAM R. WATSON.

Dr. Watson called attention to the relative infrequency of attributing aphonia to physical causes, and the prevalence of the diagnosis of functional aphonia. Acute laryngitis, tuberculosis, syphilis, benign or malignant growths, paralysis of the intrinsic laryngeal nerves following neuritis or pressure on the laryngeal nerves from any cause, hysteria, cerebral lesions affecting the cortical centers

governing laryngeal enervation and cerebral concussion, were given as the most common causes of this condition. In addition, a case of carcinoma of the right tonsillar region in a woman, aged 25, was presented. Surgical removal was accomplished with no evidence of recurrence to date.

Three illustrative cases of laryngitis were cited. In two, tuberculosis was the cause, in the third, syphilis. The first case was of especial interest, for although the laryngeal lesion was definitely shown to be tuberculous by microscopic examination, no evidence of tuberculosis elsewhere could be found.

Reference was made to Mr. Herbert Tilley's report, in 1918, of twenty-three cases of unilateral laryngeal paralysis. A follow-up of these cases showed that seven had completely regained normal function, ten were not heard from, three were unchanged, and three had died. It should be remembered that unilateral functional vocal cord paralysis can occur.

DISCUSSION

Dr. George B. Wood stated that he had seen two subjects with unilateral cord paralysis, with complete recovery.

Dr. Stauffer spoke of the prevalence of aphonia in the recent epidemic of acute colds. At the present time, he has an adult female under observation suffering from aphonia, whose larynx is normal in appearance.

Dr. James A. Babbitt cited four cases of aphonia in his practice. In one, the cause has not been determined. In another, the condition followed traumatic cellulitis of the throat. A third was due to anteropoliomyelitis limited to the laryngeal enervation. The fourth followed thyroidectomy.

Dr. George Fetterolf agreed with Dr. Watson that in primary tuberculosis of the larynx a lesion does not occur. It is comparatively easy for a small pulmonary lesion to escape detection even when our most advanced methods of examination are utilized. This lesion could caseate, break down, establish laryngeal disease, later heal and so escape detection.

Sinus Disease in Children. Presented by Dr. Charles R. Hughes (by invitation).

The method of Dr. Hughes in making a diagnosis of sinus disease in children is to make an examination of the interior of the nose by reflected light and confirm or disprove the existence of ethmoiditis. The patient is then sent to the roentgenologist whose findings are often of value. Sometimes this exposure cures a catarrhal ethmoiditis. It is of great importance to determine the presence of maxillary sinusitis. If antrum disease is present, the antrum is opened intranasally under gas. If this fails to accomplish results, a radical external operation is done.

DISCUSSION

Dr. C. C. Eves stated that the importance of sinus disease in children is becoming more recognized in recent years. The removal of tonsils and adenoids does not always clear up this condition. Exposure to the roentgen ray often helps ethmoiditis. He sometimes gives as many as five roentgen-ray treatments.

Dr. Stauffer stated that he has seen several children die of nephritis probably caused by sinus infection.

Dr. Karl M. Houser spoke of the methods of diagnosis and treatment used by Dr. Dean of Iowa City in sinus conditions of children.

Dr. Hughes was of the opinion that, in acute suppurative sinusitis complicated by nephritis, wide drainage by surgical measures should be established.

RETROPHARYNGEAL ABSCESS DISCHARGING THROUGH THE RIGHT EAR. Presented by DR. C. C. EVES.

A child, aged 20 months, was admitted to the hospital suffering from extreme dyspnea and cyanosis. A short time later the child became comfortable and a profuse discharge was noted coming from the right ear. This was later proved to be a retropharyngeal abscess that had evacuated itself through the right external auditory canal at the junction of the cartilaginous and osseous portions. The middle ear was not affected.

There are three routes this pus could have followed to reach the auditory canal:

1. Through the fissures of Santorini.

2. Through the fibrous tissue between the junction of cartilage and bone which is at times present, especially in children.

3. Through a dehiscence of the bony canal, which is present in a small percentage of cases.

Dr. Eves cited several other cases of retropharyngeal abscess that presented grave symptoms. In one, hemorrhage was sufficiently severe to require carotid ligation. In another, severe meningeal irritation was present. All patients cited recovered.

DISCUSSION

DR. GEORGE B. WOOD cited a case of quinsy which evacuated through the external auditory canal.

DR. STAUFFER mentioned another case of retropharyngeal abscess discharging through the ear.

DR. GEORGE B. WOOD mentioned a patient with quinsy, who ceased to breathe following the evacuation of a peritonsillar abscess under ether. He was resuscitated with difficulty. No pus had entered the trachea. He feels that the sudden release of pressure is in some way responsible for the failure of respiration.

DR. JAMES A. BABBITT spoke of a peritonsillar abscess in a child in whom pressure on the abscess caused failure of respiration and death. No incision had been made.

DR. GEORGE FETTEROLF had a patient cease breathing during the evacuation of a peritonsillar abscess. In this case the trouble was due to suffocation. By removing the pus from the trachea by gravity, breathing was restored.

DR. STAUFFER cited the case of an adult male who suffocated following the spontaneous evacuation of a large peritonsillar abscess.

DR. C. C. EVES called attention to the occurrence of meningeal irritation in retropharyngeal abscess. He also stated that the literature contains reports of fifty or more cases of fatal hemorrhage following the evacuation of peritonsillar abscess.